

A SCREENING STUDY ON DERMATOSES IN PREGNANCY

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CERTIFICATE

Certified that this dissertation entitled “***A SCREENING STUDY ON DERMATOSES IN PREGNANCY*** ” is a bonafide work done by **Dr.KANNAMBAL.K**, Post Graduate Student of the Department of Dermatology, Venereology and Leprosy, Madras Medical College, Chennai – 600 003, during the academic year 2008 – 2011. This work has not previously formed the basis for the award of any degree.

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CONTENTS

SI. NO.	TITLE	PAGE NO.
1	INTRODUCTION	1
2	REVIEW OF LITERATURE	2
3	AIM OF THE STUDY	38
4	MATERIALS AND METHODS	39
5	OBSERVATION	41
6	DISCUSSION	61
7	CONCLUSION	71
8	ANNEXURES	
	BIBLIOGRAPHY	
	PROFORMA	
	MASTER SHEET	

INTRODUCTION

Pregnancy is characterized by altered endocrine, metabolic, and immunologic milieus. These dramatic alterations result in multiple cutaneous changes, both physiologic and pathologic.¹ These alterations may range from normal cutaneous changes to eruptions that appear to be specifically associated with pregnancy. Moreover, pregnancy may modify the course of a number of dermatological conditions. Likewise, the concerns of the patient may range from cosmetic appearance, to the chance of recurrence of the particular problem during a subsequent pregnancy, to its potential effects on the fetus in terms of morbidity and mortality.²

The present study was undertaken to find out the prevalence of the physiological and pathological skin changes in pregnancy.

REVIEW OF LITERATURE

ENDOCRINE BACKGROUND

Pregnancy is characterized by the advent of a new and unique endocrine organ, the placenta. The hormonal changes of pregnancy start soon after the fertilized ovum becomes implanted in the endometrium, when the developing trophoblast begins to secrete chorionic gonadotrophin. This, in turn, stimulates production of estrogen and progesterone by the corpus luteum. At about the ninth week of pregnancy the fetoplacental unit begins to synthesize pregnenolone and progesterone.³ The human placenta synthesizes an enormous amount of protein and peptide hormones: lactogen (hPL), adrenocorticotropin (ACTH), growth hormone variant (hGH-v), parathyroid hormone – related protein (PTH-rp), calcitonin, relaxin, inhibins, activins, and atrial natriuretic peptide, thyrotropin – releasing hormone (TrH), gonadotropin releasing hormone (GnRH), corticotropin – releasing hormone (CRH), somatostatin and growth hormone – releasing hormone (GHRH).⁴

Steroid production rates in nonpregnant and near – term pregnant woman are shown in Table 1.⁴

Table 1:

Steroid production rates in nonpregnant and near – term pregnant woman

Steroid	Production rates (mg/24 hrs)	
	Nonpregnant	Pregnant
17 β Estradiol	0.1 – 0.6	15 – 20
Estriol	0.02 – 0.1	50 – 150
Progesterone	0.1 – 40	250 – 600
Aldosterone	0.05 – 0.1	0.250 – 0.600
Deoxycorticosterone	0.05 – 0.5	1 – 12
Cortisol	10 – 30	10 – 20

Placental hormones partly facilitate the physiological adaptations that occur in pregnancy. The pituitary enlarges and increases its output of ACTH, prolactin and gonadotropins. Circulating cortisol rises by a decrease in its clearance rate combined with increased cortisol binding globulin. The breasts enlarge during pregnancy.

The state of pregnancy is ended by an alteration in the balance of the antagonistic actions of estrogen and progesterone. This is probably fine tuned by the fetal pituitary adrenal axis and its effect on estrogen production.³

A. PHYSIOLOGICAL SKIN CHANGES RELATED TO PREGNANCY

I. Pigmentation

Pigmentary disturbances are the most common of these physiologic changes. Hyperpigmentation is seen in upto 90% of pregnant women.¹ The physiology of hyperpigmentation may be related to elevated serum levels of melanocyte stimulating hormone, estrogen, and possibly progesterone.⁵⁻⁷ Mild generalized hyperpigmentation is usually seen with accentuation of normally hyperpigmented areas such as the areolae, nipples, genital skin, axillae and inner thighs. Darkening of the skin adjoining the areolae, results in secondary areolae.⁵ Linea nigra refers to the typically reversible darkening of the linea alba, a hypopigmented linear patch extending from the pubis symphysis to the xiphoid process of the sternum.¹

Melasma (chloasma / “mask of pregnancy”) has been reported in upto 70% of pregnant women.^{5,8} This usually develops during the second half of pregnancy as irregular, sharply marginated areas of pigmentation in a roughly symmetrical pattern either on the forehead and temples, or on the central part of the face, or both.³ This type of hyperpigmentation results from excessive melanin deposition in the epidermis (70%), dermal melanophages (10% - 15%), or both (20%) and can be clinically demonstrated by wood’s lamp examination.⁵ Elevated estrogens, progesterone and MSH levels are thought to cause melasma of pregnancy.^{7,9,10} Ultraviolet and visible light may worsen melasma or allow it to persist.^{9,11} In most cases, gestational melasma will resolve postpartum. It may recur in subsequent pregnancies.⁵

II. HAIR CHANGES

Some degree of hirsutism is seen in all women, especially on the face, and less often on the arms, legs and back. This is caused by endocrine changes of pregnancy and regresses within 6 months postpartum.⁵ Although prolonged anagen phase on the scalp has been shown, the anagen to telogen ratio has varied in previous studies.^{12, 13} Other changes include a mild frontoparietal recession and diffuse

thinning in the later months of pregnancy due to inhibition of anagen hairs.⁵

III. NAIL CHANGES

It includes transverse grooving, brittleness, distal onycholysis, and subungual hyperkeratosis. The pathogenesis of nail changes is unclear.⁵

IV. GLANDULAR CHANGES

a) ECCRINE GLAND

Increased eccrine function is observed during pregnancy, except on the palms where it is diminished. This may contribute to the increased incidence of miliaria, hyperhidrosis and dyshidrotic eczema in pregnancy.^{8, 14} The increase in eccrine function has been associated with increased thyroid activity.⁵

b) APOCRINE GLAND

Apocrine activity appears to decrease during pregnancy, which may explain the clinical improvement of pre-existing Fox-Fordyce disease and hidradenitis suppurativa. The disease may rebound postpartum.⁵

c) SEBACEOUS GLAND

The rate of sebum secretion tends to increase during pregnancy and return to normal after delivery. The rise in sebum secretion during the last trimester of pregnancy is due to a powerful sebotropic stimulus released from pituitary.¹⁵ Secretion of pituitary factors, such as prolactin, which either stimulate sebaceous glands directly or enhance their response to androgens.³ The sebaceous glands on the areolae enlarge and appear as small brown papules called Montgomery's glands or tubercles. The effect of acne is unpredictable, but in many patients acne develops for the first time during pregnancy.⁵

V. CONNECTIVE TISSUE CHANGES

Striae distensae (Striae gravidarum) develop in upto 90% of women during the sixth and seventh months of pregnancy. They appear as pink or purple atrophic bands on the abdomen and sometimes on the breasts, thighs, and inguinal areas.⁵ Risk factors include genetic predisposition, family history, race.¹ Hormonal: adrenocortical hormones, estrogen, relaxin and physical factors¹⁶ – (stretching secondary to increase in the abdominal girth) play a role in the development of the striae. There seems to be an association between maternal weight gain,

fetal birth weight and the development of striae.⁵ Striae are commonly absent in pregnancy in Ehlers Danlos syndrome.³

Molluscum fibrosum gravidarum (skin tags) often appear in the second half of pregnancy probably due to hormonal factors.³

V. VASCULAR CHANGES

Vascular changes are thought to be due to sustained high levels of circulating estrogen resulting in distension and proliferation of vessels.³ The changes include spider telangiectases, palmar erythema, nonpitting edema, varicosities, vasomotor instability & purpura. Spider telangiectases are observed in areas drained by the superior vena cava, such as around the eyes. These are seen in 67% of white patients, appear between the second & fifth month of pregnancy. Palmar erythema is seen as a diffuse pink mottling of the whole palm, or may be confined to the thenar and hypothenar eminences.⁵ 5% of pregnant women may develop small hemangiomas.³

Nonpitting edema of the face, eyelids, and extremities is observed in at least half of all pregnant women. The swelling is most pronounced in the early morning and disappears during the course of the day.⁵ Varicosities appear in 40% of patients and result from increased venous

pressures in the femoral and pelvic vessels caused by the gravid uterus.¹⁷ Vasomotor instability manifested as facial flushing, pallor, hot and cold sensations, cutis marmorata of the legs, worsening of preexisting raynaud's phenomenon, dermographism and urticaria are common during pregnancy. Purpuric lesions are common on the legs in the second half of pregnancy.⁵

VI. MUCOSAL CHANGES

Hyperemia of the gums is seen in 80 percent or more of pregnant women with varying degrees of severity and may be associated with gingivitis.^{18, 19} It develops in third trimester of pregnancy and resolves postpartum. Proliferation of capillaries within the hypertrophied gingiva results in granuloma gravidarum, pyogenic granuloma or pregnancy epulis that appears between the second and fifth months of pregnancy in 2% of pregnant women. It is seen as a deep – red nodule on the buccal or lingual surface of the marginal gingiva.⁵ Increase in vascularity of lesion is due to high estrogen levels.³

B. DERMATOSES OF PREGNANCY

Dermatoses of pregnancy include conditions that occur exclusively during pregnancy and results directly from the state of gestation or the products of conception.

Currently, two main classification schemes are used. The first, proposed by Holmes and Black in 1983, include pemphigoid gestationis, polymorphic eruption of pregnancy, prurigo of pregnancy and pruritic folliculitis of pregnancy. The second proposed by Shornick in 1998, consists of – pemphigoid gestationis, polymorphic eruption of pregnancy, prurigo of pregnancy and intrahepatic cholestasis of pregnancy. He suggested that pruritic folliculitis and papular dermatitis belong to the group of prurigo of pregnancy.²⁰ Moreover, impetigo herpetiformis is believed to be a variant of pustular psoriasis that is triggered by pregnancy rather than a specific dermatosis of pregnancy.²¹

I. INTRAHEPATIC CHOLESTASIS OF PREGNANCY (ICP)

Synonyms: Obstetric cholestasis, idiopathic jaundice of pregnancy, prurigo gravidarum, pruritus gravidarum, and icterus gravidarum. It was first recognized by Svanborg and Ohlsson.¹ The incidence varies from 0.02 to 2.4%.²² It may be subdivided according to those patients with hyperbilirubinemia (cholestatic jaundice of pregnancy) and those with pruritus and biochemical abnormalities but without hyperbilirubinemia (prurigo gravidarum).^{23,24}

Pathogenesis

i) Estrogens, such as estriol-16 α D-glucoronide and estradiol 17 β glucoronide reduces the sodium dependent bile acid uptake into the hepatocyte and inhibits basolateral transport proteins.²⁵ Furthermore, the increased levels of sulfated progesterone metabolites in the serum may saturate the maximal transport capacity of membrane transport proteins of the hepatocyte.²⁶ Hormonal etiology is probable because ICP is a disease of late pregnancy corresponding to the period of highest placental hormone levels, and spontaneously remits at delivery when hormone concentrations normalize, and it recurs during subsequent pregnancies in 45 to 70 percent of patients.¹ ii) Geographical variation and familial clustering indicate a genetic predisposition. Candidate genes include ABCB4 (multidrug resistance gene 3), ABCB11 and ATP8B1.¹ Heterozygosity for deletion in the multidrug resistance gene 3 was found in women with ICP.²¹ iii) Reports of higher incidence of ICP with reduced selenium levels, hepatitis C virus infection and during winter months point towards the role of environmental factors.²⁷ High bile acid concentration in the serum and skin causes pruritus and it correlates with the intensity of pruritus.²²

Clinical features

It usually manifests in the third trimester. The disorder has no primary cutaneous lesions, although secondary excoriations may occur.²² There is a family history in 50% of cases and an association with multiple gestation.²¹ Patients present with severe pruritus which is more during the night time, initially localized over abdomen and extremities and then becomes generalized. It may precede jaundice by atleast 4 weeks. Fatigue, anorexia, nausea, vomiting and icterus may be seen. Right quadrant fullness, dark urine and light colored stools may be present. Nowadays anicteric cases with itching should be regarded as merely a clinical variant.²² This condition resolves within the first month after delivery.²¹

Laboratory studies

Reveals elevated lactate dehydrogenase, alkaline phosphatase, serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT) and bilirubin levels. Postprandial bile acid levels are regarded as a very sensitive indicator of the disease.²² Cholic acid – chenodeoxycholic acid ratio is greater than 1.5.¹

Maternal complications

Postpartum hemorrhage and gallstones.²⁷

Fetal complications

Includes perinatal death, prematurity, low birth weight.²⁷

Decreased fetal elimination of toxic bile acids may cause vasoconstriction of placental chorionic veins and meconium passage.²⁸ Meconium can cause acute umbilical vein constriction resulting in asphyxia.²¹

Treatment

Antipruritics and emollients in mild cases. Cholestyramine and ursodeoxycholic acid in severe cases.

II. PRURITIC URTICARIAL PAPULES AND PLAQUES OF PREGNANCY (PUPPP)

Synonyms: Polymorphic eruption of pregnancy, Bourne's toxemic rash of pregnancy, Nurse's late prurigo of pregnancy and toxic erythema of pregnancy. Its incidence ranges between 1 in 130 pregnancies and 1 in 300 pregnancies. The term PUPPP was coined by Lawley et al (1979).¹

Pathogenesis

The pathogenesis of PUPPP is uncertain. A role for sex hormones has been suggested.²¹ A hypothesis suggested that during the aging of placenta in the third trimester, a substance is released into the maternal circulation that may trigger fibroblast proliferation.⁵ Several authors suggested that rapid abdominal wall distension in primigravidas may cause damage to connective tissue in the striae with subsequent conversion of nonantigenic molecules to antigenic ones, thus triggering an inflammatory process.²⁹⁻³² There is an association of PUPPP with multiple gestation.³³ Multiple gestation is associated with excessive abdominal distension, it may cause trauma to the skin triggering an inflammatory reaction. Furthermore, multiple gestation is associated with higher estrogen and progesterone levels.³⁴

Progesterone has been shown to aggravate the inflammatory process at tissue level, and increased progesterone receptor immunoreactivity has been detected in skin lesions of PUPPP.³⁵ Increased abdominal stretching increases vascular permeability and, hence may facilitate the migration of fetal chimeric cells into the maternal skin. Familial occurrence has been reported.²¹

Clinical features

PUPPP occurs predominantly in primigravidas in the third trimester and does not usually recur in subsequent pregnancies. The eruption is intensely itchy, polymorphous, showing urticarial and at times, vesicular, purpuric, polycyclic or targetoid lesions. The lesions start in the abdominal striae and shows periumbilical sparing. The rash then spreads over a few days to involve the thighs, buttocks, breasts and arms. The face, palms and soles are spared.²¹ The mean duration of eruption is 6 weeks. Within an individual patient, lesions are generally morphologically uniform. Spontaneous remission within days of delivery is the rule.¹

Laboratory investigations

Histopathology shows spongiotic dermatitis with perivascular or upper dermal inflammatory cell infiltrate. There may be a striking number of eosinophils.⁵

Maternal complications : Not seen

Fetal complications : Not seen

Treatment

Symptomatic with antipruritics and topical corticosteroids.

III. PEMPHIGOID GESTATIONIS (PG)

This is a rare, self-limiting blistering dermatosis of pregnancy with an incidence of 1 in 50,000 pregnancies. The term pemphigoid gestationis was proposed in 1982 by Holmes and Black.³⁶

Pathogenesis

Pemphigoid gestationis is an autoimmune disease. The main antigenic determinant of PG is collagen XVII (BPAG2 or BP 180), found in both the basement membrane zone (BMZ) of skin and the amniotic epithelium of the placenta and the umbilical cord.³⁷⁻³⁹ The antigenic epitopes are restricted within the noncollagenous16A (NC16A) region.^{40, 41} It is believed that an aberrant expression of MHC class II molecules in the placenta leads to the presentation of this self antigen and the triggering of an immune response with antibody production. PG antibodies are IgG, mainly IgG1 and IgG3. The antibodies cross react with collagen XVII in the skin, form immune complexes, activate complement and recruit inflammatory cells. The inflammatory mediators released by various cells in the infiltrate induce

tissue damage and blister formation.⁴² The presence of a serum PG antibody was first described by Provost and Tomasi known as HG factor. The development of PG is genetically predetermined by the presence of MHC class II HLA antigens DR3 and DR4. There is the presence of anti HLA antibodies against paternal HLA molecules in virtually all PG patients. These antibodies are primarily against HLA class I antigens and are separate from circulating anti BMZ antibodies.⁴³ There is a strong hormonal association. Estrogen has immune enhancing properties by increasing the B cell response, with resultant stimulatory effect on PG.⁴⁴

Clinical features

PG usually develops in the second or third trimester of pregnancy with a mean onset of 21-28 weeks gestation. Onset is with severe pruritus followed by erythematous urticarial papules and plaques that may become target like or polycyclic. Within days to weeks, progression occurs with the formation of clustered tense vesicles and bullae at the sites of urticated erythema. Resolution occurs without scarring. In about 50-90%, the eruption begins on the abdomen periumbilically or within the umbilicus and later spreads to involve the whole abdomen, extremities, palms and soles. Face and mucous membranes are spared. Spontaneous remission after parturition occurs and tends to recur in the

subsequent pregnancy. PG has been reported as a paraneoplastic presentation in association with hydatidiform mole and choriocarcinoma.³⁶

Laboratory investigations

Histopathology shows eosinophilic spongiosis, papillary dermal edema, perivascular infiltrate composed of lymphocytes and eosinophils. Focal necrosis of basal keratinocytes leads to a subepidermal blister.⁴⁵ Direct immunofluorescence shows a heavy linear deposition of C3 and IgG along the BMZ of skin, BMZ of amniotic epithelium and in the skin of newborns of affected mothers. Indirect immunofluorescence detects C3 and IgG in the sera.³⁶ In salt split skin specimens, the antibody binds to the roof of the vesicle.⁵

Maternal complication: Premature delivery, spontaneous abortion.

Fetal complication: Small for gestational age⁴⁶, neonatal disease with blisters due to passive transfer of immunoglobulins.⁴⁷

Treatment

For mild cases - topical corticosteroids and oral antihistamines. Severe cases require systemic corticosteroids.

IV. PRURIGO OF PREGNANCY (PP)

Prurigo of pregnancy comprises the previously described Besnier's prurigo gestationis and Nurse's early prurigo of pregnancy, papular dermatitis of Spangler.⁵ Incidence is 1 in 300 to 450 pregnancies.²¹

Aetiopathogenesis

The cause is unknown. The condition may be related to an atopic background or ICP or both. Serum IgE levels are elevated which is consistent with the theory that PP arises as a result of pruritus gravidarum in women with an atopic predisposition.⁵

Clinical features

Onset is usually in the second or third trimester. Lesions consist of pruritic, erythematous, excoriated or crusted papules over the extensor surfaces of the extremities, occasionally on the abdomen.⁴⁸ It resolves after delivery. Recurrence during subsequent pregnancy is common.⁴⁹

Laboratory investigation

Histopathology is nonspecific with chronic inflammatory cell infiltrate in upper dermis with occasional epidermal changes.⁵

Maternal complications : None

Fetal complications : None

Treatment : Symptomatic

V. PRURITIC FOLLICULITIS OF PREGNANCY (PFP)

PFP was described by Zoberman and Farmer in 1981.⁵⁰ The incidence is 1 in 3000 pregnancies.¹

Aetiology

Unknown. Wilkinson et al postulated that PFP is related to hormonal changes in pregnancy.⁵⁰

Clinical features

Onset is during the second or third trimester, and spontaneous resolution occurs within 2 to 3 weeks postpartum. Recurrence in subsequent pregnancies is uncommon.¹ The lesions are pruritic, follicular, erythematous, monomorphous papules and pustules over trunk.⁵⁰

Laboratory diagnosis

Histopathology reveals sterile folliculitis.⁵⁰

Maternal complications : None

Fetal complications : Decreased birth weight

Treatment : Symptomatic

VI. ATOPIC ERUPTION OF PREGNANCY

Ambros – Rudolph et al have proposed the term atopic eruption of pregnancy to denote a new disease complex comprising the previously distinct entities PP and PFP as well as eczema in pregnancy.²⁰ The features include onset early in pregnancy, a predilection for flexural skin, personal and/or family history of atopy, skin lesions with either eczematous features (E type AEP) or prurigo type lesions (P type AEP), flare up of preexisting atopic dermatitis. Total serum IgE levels may be elevated. No specific association with parity or gestation.¹ Th2 shift associated with pregnancy may favour the exacerbation of atopic dermatitis and the manifestation of AEP.²⁰

C. DERMATOSES MODIFIED BY PREGNANCY

I. INFECTIONS

The increased incidence of certain infections in pregnancy has been attributed to the immunosuppressive effects of high serum levels of

estrogen. These include a decrease in cell mediated immunity, neutrophil function, and activity of natural killer cells, as well as impairment of local antibody responses.³

1. CANDIDIASIS

Candida albicans exists as a commensal organism in the vaginal mucosa of upto 30% of pregnant woman.¹ Higher glycogen content in the vaginal environment and estrogen mediated enhanced adherence of *Candida* species to vaginal epithelial cells results in an increased risk of symptomatic vaginitis in pregnancy.⁵¹ There is vulval pruritus with cottage cheese like vaginal discharge and vaginal soreness. Diagnosis is confirmed by 10% KOH examination of discharge and culture.

2. DERMATOPHYTOSIS

The fungus invades the stratum corneum possibly aided by warm occlusive conditions. Incidence of dermatophytosis during pregnancy is similar to that in normal individuals. Typical lesion is annular with erythematous scaly margin and central clearing. Diagnosis is by scraping and examination in 10% KOH and culture.

3. PITYROSPORUM FOLLICULITIS

Pityrosporum folliculitis caused by the yeast *Malassezia furfur* occurs with greater frequency in pregnant women.⁵² The lesions are itchy follicular papules over the shoulder and back.

4. PITYRIASIS VERSICOLOR

Pregnancy may have some influence in increasing the susceptibility but firm data is lacking.⁵³ Lesions are well defined macules with fine branny scaling over seborrhoeic areas. Diagnosis is by scraping examination in 10% KOH.

5. LEPROSY

Altered course of leprosy in pregnancy are due to

- i) Metabolic and immunologic changes during pregnancy
- ii) Altered secretion of steroids, as the levels of free cortisol and 17-hydroxy corticosteroid increase during pregnancy and may partly be responsible for the depression of CMI.⁵⁴

Effects of pregnancy on woman with leprosy

1) Worsening of leprosy

- a) Women already infected with *Mycobacterium leprae* and incubating leprosy are likely to show overt signs of the disease in pregnancy.
- b) With established leprosy the disease worsens during pregnancy. The changes are due to depression of cell mediated immunity during pregnancy. The possible reason is increase in circulating thyroxine (T4) during pregnancy.⁵⁵

2) Increased incidence of lepra reactions

a) Type I reaction

During pregnancy, downgrading reaction may occur because of decreased cell mediated immunity, and is most likely to be manifested in the III trimester. In borderline leprosy, upgrading reactions are most likely to occur during the puerperium.⁵⁵

b) Type 2 reaction (Erythema nodosum leprosum reaction)

In lepromatous leprosy, this type of reaction is most likely to occur in the III trimester of pregnancy.⁵⁵

Effect on the infant

- 1) Low birth weight babies.⁵⁶
- 2) The infant has higher risk of contracting leprosy from a) the mother, if she is an open case b) hidden source of infection in the home among relatives c) other patients if born in a leprosarium.⁵⁵

6. CONDYLOMA ACUMINATA

The prevalence of HPV infection in pregnancy is 5 to 15%.⁵⁷ The genital warts frequently increase in size and number during pregnancy, sometimes filling the entire vaginal canal or covering the perineum thereby making vaginal delivery or episiotomy difficult.⁵¹ Certainly the moistness offered by vaginal mucus throughout pregnancy offers ideal moist conditions for viral growth. Accelerated viral replication with advancing pregnancy has been hypothesized to explain the growth of perineal lesion, progression of some to cervical neoplasm, and the increased detection of viral DNA from the cervix of pregnant women. The lesions regress rapidly after delivery.⁵⁷ Fetal complications include vertical transmission, juvenile laryngeal papillomatosis.

7. HERPES SIMPLEX

Effect on pregnancy:

80% of young women with recently acquired genital herpes infection will have an average of 2 to 4 symptomatic recurrences during pregnancy. 10% of recurrences in pregnancy are asymptomatic and these are more frequently around the perineum than the cervix.⁵⁷ Primary episode during pregnancy may be more severe than in nonpregnant women.⁵¹ Primary HSV infection leads to grouped vesicular lesions. Severe disease may present with gingivostomatitis and vulvovaginitis herpetica and dissemination. Risk of neonatal infection is as low as 1% if mother acquires genital herpes in first trimester, due to formation of protective antibodies, whereas the risk significantly rises to 30 – 50% if mother gets infected in last trimester.⁵⁸ Diagnosis is by viral culture, DIF and PCR. ACOG guidelines recommend that caesarian delivery is indicated only if the active lesions are present at the time of labour or within 4 – 6 hours of rupture of the membranes.⁵¹

Maternal complication: Spontaneous abortion, preterm labour

Fetal Complication: Intrauterine growth retardation, congenital herpes, neonatal herpes – (disseminated / localized / asymptomatic).

8. VARICELLA ZOSTER

Effect on Pregnancy:

Maternal varicella in the first 20 weeks of pregnancy is associated with an approximate 2% risk of fetal damage, including skin lesions, CNS and ocular defects, and limb hypoplasia, with a 30% mortality within the first year of life.⁵⁹ Maternal zoster in pregnancy is not associated with intrauterine infection.⁶⁰ Zoster in infancy has followed maternal varicella, the baby's primary infection having occurred in utero. If the mother has varicella within 4 days before to 2 days after delivery, the neonate would have no maternal antibody and is at risk of severe varicella with a mortality rate upto 30% in the absence of treatment.⁵⁹

9. HUMAN IMMUNODEFICIENCY VIRUS

The prevalence of HIV infection in pregnant women in India is 0.3%.⁶¹

i) Effects of pregnancy on HIV

Pregnancy does not alter disease progression in asymptomatic women and those with early disease, although there may be a more rapid progression in women with late stage HIV infection.⁶¹

ii) Effects of HIV on Pregnancy

Spontaneous abortion, ectopic pregnancy, preterm labour, stillbirths, low birth weight babies and opportunistic infections.⁶¹

iii) Mother to child transmission

The maximum transmission occurs in late pregnancy and during labour. The total risk of transmission is 25 – 40%. The distribution risk is as follows: Antepartum: 10 – 15%, intrapartum: 65 – 75%, postpartum: 10 – 15%. Maternal viral load is the strongest predictor of vertical transmission.⁶¹

10. TRICHOMONIASIS

Trichomonas is seen in upto 60% of pregnant women but has no adverse effects on the fetus.⁵ Trichomonal vaginitis is characterized by yellow vaginal discharge, abnormal odour, vulval pruritus, vulvovaginal erythema and colpitis macularis or strawberry cervix. Trichomonads are identified in a wet mount of vaginal secretion as flagellated, ovoid, motile organism. There is an increased risk of preterm delivery, premature rupture of membranes and postpartum fever. Female neonates may acquire infection perinatally but this seems to be transient due to maternal estrogenic influence.⁵⁷

II. AUTOIMMUNE DISORDERS

1) SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)

a) Pregnancy impact on the disease.

Cutaneous flares are the most common manifestation of SLE in pregnancy, followed by arthritis. Painful vasculitic lesions on the extremities are the most common skin lesions.⁵ Worsening of SLE is uncommon, if the individual is on immunosuppressive therapy.⁶² If conception occurs during the active stage of SLE, approximately 50% of patients will worsen during pregnancy, and a few will die or experience permanent renal damage.⁵ Complement activation is associated with disease flares during pregnancy, and measures of complement C4 and CH 50 will decrease during flares.⁶³

b) Disease impact on pregnancy

Clinical remission for 6 months before conception should indicate an uncomplicated pregnancy and a livebirth. There is a higher risk of premature delivery and fetal loss (abortion in 8% and perinatal mortality in 13%). The increased risk of fetal death is due to immune complex deposition on the trophoblast basement membrane, or the transplacental passage of antiphospholipid antibodies.⁶² The antiphospholipid antibody syndrome present with recurrent miscarriage, thrombosis, livedo reticularis, migraine, stroke, and / or thrombocytopenia.⁶⁴

2) SYSTEMIC SCLEROSIS

a) Effect of pregnancy on disease

Raynaud's phenomenon usually improves during pregnancy, particularly with the increased cardiac output in the second half of pregnancy. Gastroesophageal reflux worsens in pregnancy.^{65, 66} Early rapidly progressing diffuse systemic sclerosis are at greatest risk for developing serious cardiopulmonary and renal problems during pregnancy. The greatest danger in pregnancy is the occurrence of renal crisis, secondary to acute onset severe hypertension.⁶⁵

b) Effect of disease on pregnancy

Preterm births and small full term neonates.⁶⁵

3) DERMATOMYOSITIS AND POLYMYOSITIS

a) Effect of pregnancy on disease

Pregnancy exacerbates the disease in approximately 50% and is associated with remission in 20%.⁶⁷ Classic dermatomyositis (DM) and clinically amyopathic dermatomyositis (CADM) can occur for the first time in pregnancy. Pregnancy can be a complicating factor for preexisting classic DM and CADM. DM presenting in the first trimester of pregnancy

worsens maternal health and fetal outcome. The impact of pregnancy on DM is thought to be a function of DM disease activity and maternal overall health status.⁶⁸

b) Effect of disease on pregnancy

Women in remission at the time of conception tend to have a more favourable outcome.⁶⁹ Spontaneous abortion, intrauterine growth restriction, intrauterine death and premature delivery are common.⁷⁰

III. METABOLIC DISORDERS

1. PORPHYRIA CUTANEA TARDA

Pregnancy and the postpartum period, as well as estrogen-containing birth control pills, have been reported to exacerbate this condition. Moreover, intake of iron supplements during pregnancy may trigger the appearance of PCT.⁷¹

2. ACRODERMATITIS ENTEROPATHICA

Exacerbation during pregnancy appears to be characteristic in some patients. Pregnancy may cause a decrease in zinc concentration, however this fall in zinc levels may not be entirely due to increased fetal demand for zinc, estrogen may also play a role. Spontaneous remission occurs postpartum.⁷¹

IV. CONNECTIVE TISSUE DISEASES

1) PSEUDOXANTHOMA ELASTICUM (PXE)

Disease exacerbates during the course of pregnancy, with the risk of exacerbation of cutaneous, vascular and ocular lesions. The main complication is gastrointestinal hemorrhage. Worsening of lesions occurs in abdomen due to its greatest distension. Other risks include arterial hypertension, thromboembolic phenomena and periumbilical perforating PXE that seem to increase after multiple gestation.⁷²

Fetal risk: Abortions, intrauterine growth retardation, risk of genetic transmission of the disease to the child.⁷²

2. EHLERS – DANLOS SYNDROME

During pregnancy: The aggravation of the symptoms, mainly in articulations, are being reported. The reports of premature births and abortions appeared to be a result of cervical insufficiency during the first and second trimesters of pregnancy or, also to a spontaneous rupture of the fetal membrane. Other complications include hemorrhage, difficult cicatrization with dehiscence of the suture, hernias and hematomas. Form IV or arterial presents risk of death to the mother that may reach 25%, mainly by the rupture of the aorta or uterus. These complications seem to be worse during labour and in the early postpartum period. Fetal risk includes retarded growth, hernia, luxation and floppy infant syndrome.⁷²

3) ANETODERMA

During pregnancy: It is likely that some correlation exists between the modification of elastic tissue occurring in pregnancy and appearance of anetoderma. As antiphospholipid antibodies and systemic lupus are associated with anetoderma, it would be advisable to request phospholipid antibodies investigation for lupus in pregnant women who have developed a picture of primary anetoderma.⁷²

V. CUTANEOUS TUMORS

The association of pregnancy with hemangioma, hemangioendothelioma, and glomus tumor reflects the effect of gestational hormones on the vascular structures. Large hemangiomas resulting in arteriovenous shunting and high output cardiac failure have been reported, these lesions regressed postpartum. New glomangiomas may occur in successive pregnancies.⁵

Dermatofibromas, leiomyomas, desmoid tumors and keloids may develop or grow rapidly during pregnancy. Neurofibromas may enlarge or arise denovo during pregnancy, plexiform neurofibromas may enlarge rapidly. This is often associated with massive hemorrhage within the tumor. Patients may experience severe vascular complications during pregnancy such as hypertension and renal artery rupture. In most cases, a partial regression of the lesion occurred postpartum.⁵

The relationship between pregnancy and malignant melanoma has been debated.⁵ Epidemiologic studies from the USA have failed to show significant associations between melanoma and reproductive and other hormonal factors in women.³ Grin, Driscoll, and Gromtkels reviewed controlled clinical trials to assess the effect of pregnancy on the prognosis of melanoma and showed that pregnancy does not influence the 5 year survival rates.⁷³ Pregnancy may exacerbate mycosis fungoides and the eosinophilic granuloma form of langerhans cell histiocytosis.³

VI. MISCELLANEOUS DISORDERS

1. ACNE VULGARIS

Acne may improve during pregnancy, but it is occasionally exacerbated during pregnancy. This is particularly the case with acne conglobata.⁷⁴

2. ERYTHEMA MULTIFORME

Pregnancy may trigger erythema multiforme. There are reports of vaginal stenosis resulting from Stevens - Johnson syndrome occurring in pregnancy.⁷⁴

3. URTICARIA

Urticaria, particularly physical – pressure type, can occur during pregnancy and is often aggravated by pregnancy. It appears mainly on the abdomen in the second but more in the third trimester. The cause is unclear but precipitating factors may be heat, constricting clothing, or even the distension of the abdominal wall.⁷⁴

4. PSORIASIS

The effects of pregnancy on psoriasis are variable. Psoriasis is more likely to improve during pregnancy with relapse postpartum. Pregnancy is associated with an increase in the levels of estrogen and progesterone, which may play a role in improving psoriasis by promoting a state of immune tolerance. It acts in stimulation of B cell mediated immunity but suppresses the T cell mediated immunity.^{74,75} Progesterone has also been shown to be the key factor in uterine immunosuppression.^{76,77}

Clinically, chronic plaque psoriasis is the most common type of psoriasis to develop or worsen in pregnancy. In general, chronic plaque psoriasis is more likely to improve (40% - 63%) than worsen (14%), due to high levels of interleukin 10 in pregnancy.⁷⁴

5. IMPETIGO HERPETIFORMIS

It is a variant of generalized pustular psoriasis seen in pregnancy. It was first described by Von Hebra in 1872.⁷⁸

Aetiopathogenesis

An endocrine cause is suspected. The worsening of pustular psoriasis just before menstruation and challenge with progesterone and clomiphene produces pustular exacerbations in such patients.³

Clinical features

Onset is usually in the last trimester of pregnancy. It presents as symmetric, erythematous patches with grouped pustules at their margins. The lesions start in the intertriginous or flexural areas and extend centrifugally. Condyloma like lesions may form in the flexures. Subungual pustules may cause onycholysis.⁵ Tongue, buccal mucosa and esophagus may be involved. Constitutional disturbances can occur.³ Remission is seen postpartum, but recurrence in subsequent pregnancies may occur.⁷⁹

Laboratory investigation

Leukocytosis, elevated ESR, decreased serum calcium, vitamin D and albumin may be found.⁵ Histopathology shows spongiform macropustules of kogoj. Parakeratosis and elongation of rete ridges are often found.⁸⁰ Culture of pustule is sterile.

Maternal Complications : Tetany, seizures and delirium.⁵

Fetal complications : Placental insufficiency leads to stillbirth, neonatal death or fetal abnormalities.⁷⁹

Treatment

Systemic corticosteroids are the mainstay of therapy.¹

6. ERYTHROKERATODERMA VARIABILIS

Marked deterioration in erythrokeratoderma variabilis occurred during pregnancy in two related women.³

AIM OF THE STUDY

1. To study the prevalence of physiological cutaneous changes in pregnant women.
2. To study the prevalence of specific dermatoses of pregnancy.
3. To study the prevalence of various infective and miscellaneous cutaneous diseases in pregnancy.
4. To correlate the prevalence of the major cutaneous changes and diseases in relation to different trimesters of pregnancy and with gravidity.

MATERIALS AND METHODS

Study design: A cross-sectional study was conducted during the period of August 2008 to August 2010. Ethical committee clearance was obtained.

Study sample: Five hundred pregnant women who attended the outpatient department of Maternity Hospital, Institute of Obstetrics and Gynaecology (Egmore), Chennai were randomly selected, irrespective of the duration of pregnancy and gravidity.

Informed consent was obtained before clinical examination. Detailed history including age, obstetric status, antenatal history, chief complaints related to skin, onset of skin changes / lesions in relation to duration of pregnancy, history of similar illness in previous pregnancies, family history of similar lesions, exacerbating factors, associated medical and skin diseases, etc were elicited and recorded.

Complete dermatological examination was done in all cases to study the physiological and pathological changes of skin and its appendages. If any specific dermatosis of pregnancy was present, the morphology of skin lesions, distribution and the sites involved were studied. Relevant systemic examination was carried out. If any

preexisting skin disease was present, evidence of exacerbation or remission was recorded.

Appropriate investigations were done if required to confirm the diagnosis. Bedside laboratory procedures like KOH mount, Gram's stain and Tzanck smear were carried out. To confirm diagnosis skin biopsy was done in a few cases. In all cases with history of pruritus related to specific disorders of pregnancy, liver function tests were done. Screening with VDRL and ELISA for HIV was done in all cases. Examination of the contact was done in all cases of sexually transmitted disease. Results were tabulated and analyzed. Statistical analysis of major cutaneous changes between primigravidas and multigravidas, and between various trimesters were done by Fisher's exact test and Chi square test respectively.

OBSERVATION

A total of 500 pregnant women were studied from August 2008 to August 2010. Of these, 291 (58.2%) were primigravidas, 209 (41.8%) were multigravidas. Among the multigravidas, 164 (32.8%) were second gravida , 39 (7.8%) were third gravida , and 6 (1.2%) were fourth gravid. Cases seen in the I trimester were 9 (1.8%), II trimester were 87 (17.4%) and III trimester were 404 (80.8%). 98.2% were in the II and III trimester of pregnancy.

Distribution of cases among various gravidas and trimesters is shown in Table 1(**Fig 1a&1b**).

Table 1

Distribution of cases among various gravidas and trimesters (n=500)								
Parity	I Trimester		II Trimester		III Trimester		Total	
	No of Pregnant women	Percentage	No of Pregnant women	Percentage	No of Pregnant women	Percentage	No of Pregnant women	Percentage
Gravida -1	2	0.4	33	6.6	256	51.2	291	58.2
Gravida -2	5	1	41	8.2	118	23.6	164	32.8
Gravida -3	2	0.4	8	1.6	29	5.8	39	7.8
Gravida -4	0	0	5	1	1	0.2	6	1.2
Total	9	1.8	87	17.4	404	80.8	500	100

STUDY POPULATION CHARACTERSTISTICS

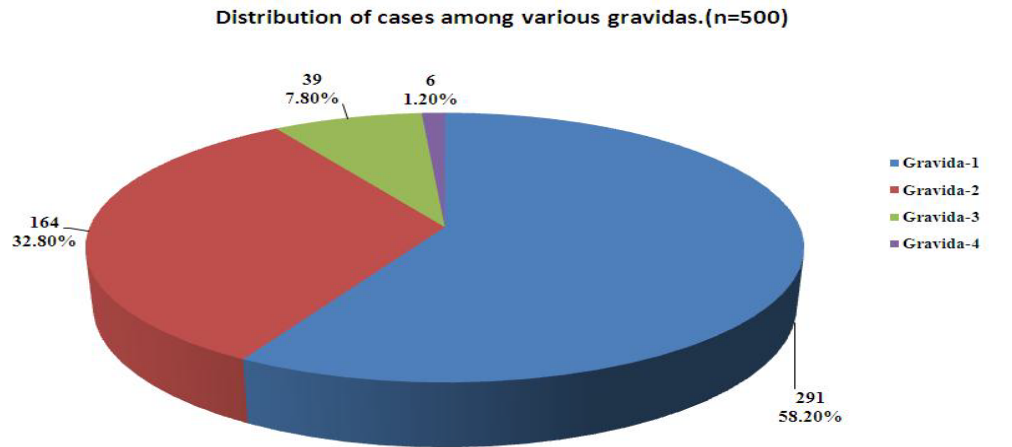


Fig 1a

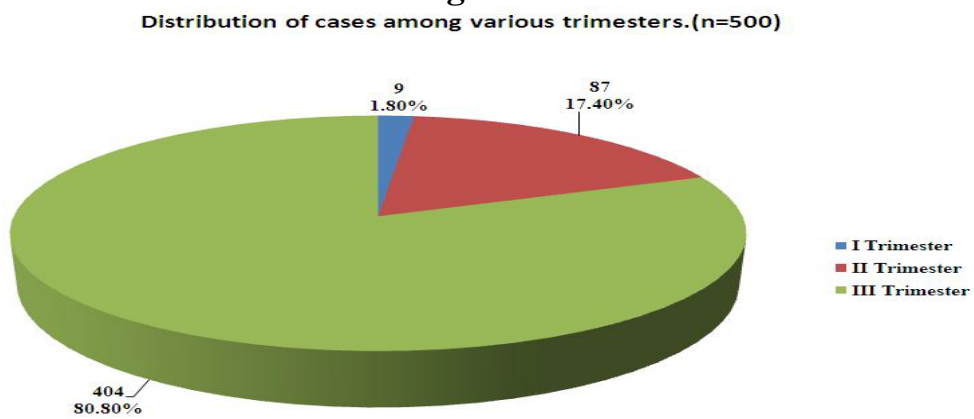


Fig 1b

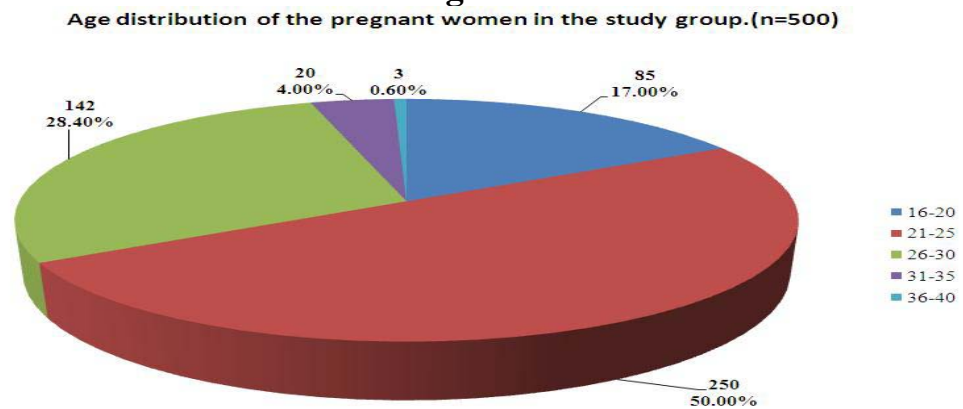


Fig 1c

Age distribution of the pregnant women in the study group is shown in Table 2 (**Fig 1c**).

Table 2

Age distribution of the pregnant women in the study group.(n=500)

Age Group in Years	Total Number	Percentage
16-20	85	17.00%
21-25	250	50.00%
26-30	142	28.40%
31-35	20	4.00%
36-40	3	0.60%

The youngest and oldest patients were aged 16 and 39 respectively. 50% (n=250) of them belonged to the age group 21-25 years. Majority of the cases had type IV and type V skin. Most of them belonged to lower socioeconomic status.

The distribution of physiological changes observed during pregnancy (n = 500) is shown in Table 3 (**Fig 2a**).

The distribution of physiological changes among various gravidas is shown in **Fig 2b**.

The distribution of physiological changes among various trimesters is shown in **Fig 2c**.

Fig 2a. Distribution of physiological changes observed during pregnancy

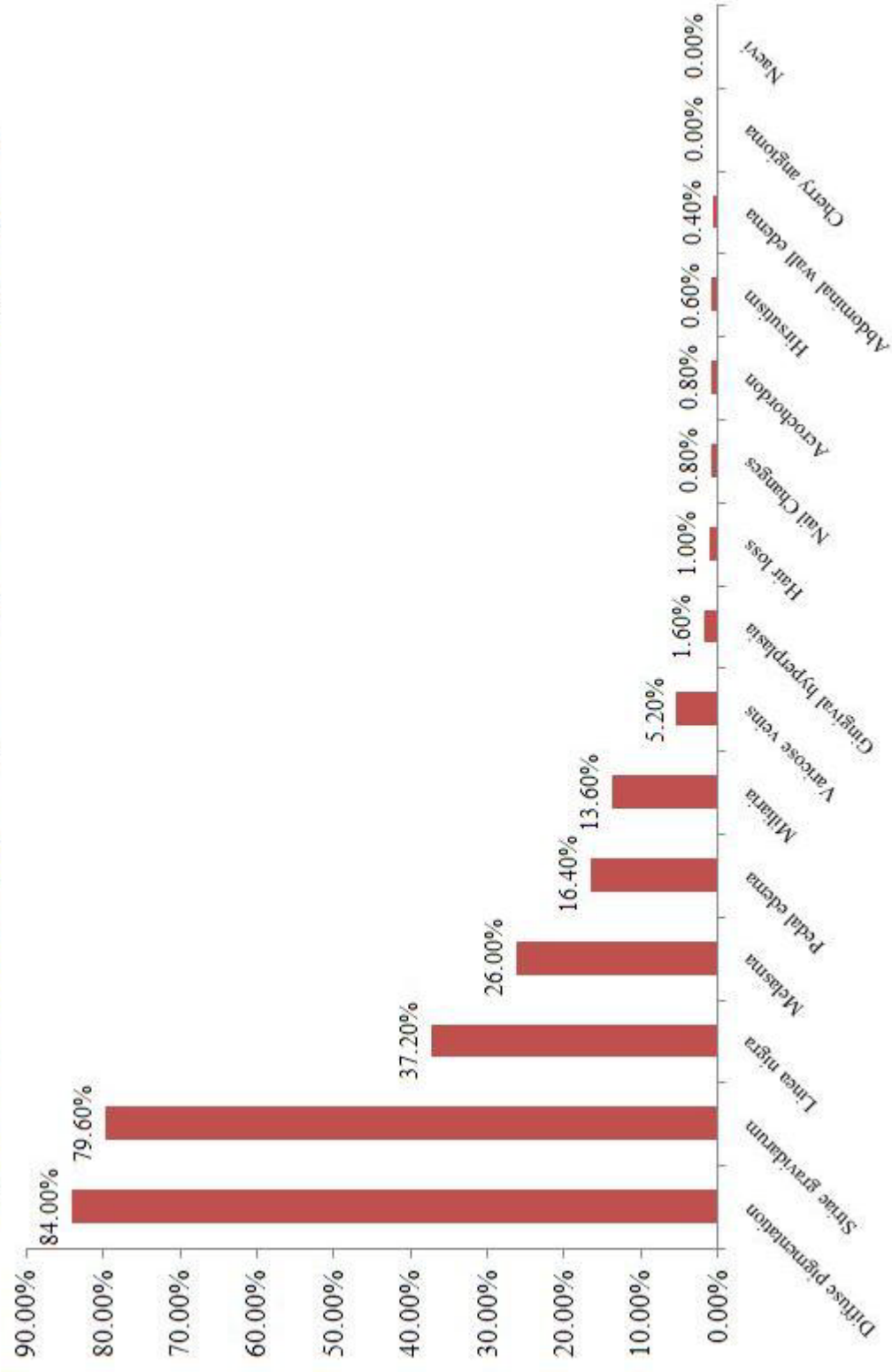


Fig 2b. Distribution of physiological changes among various gravidas

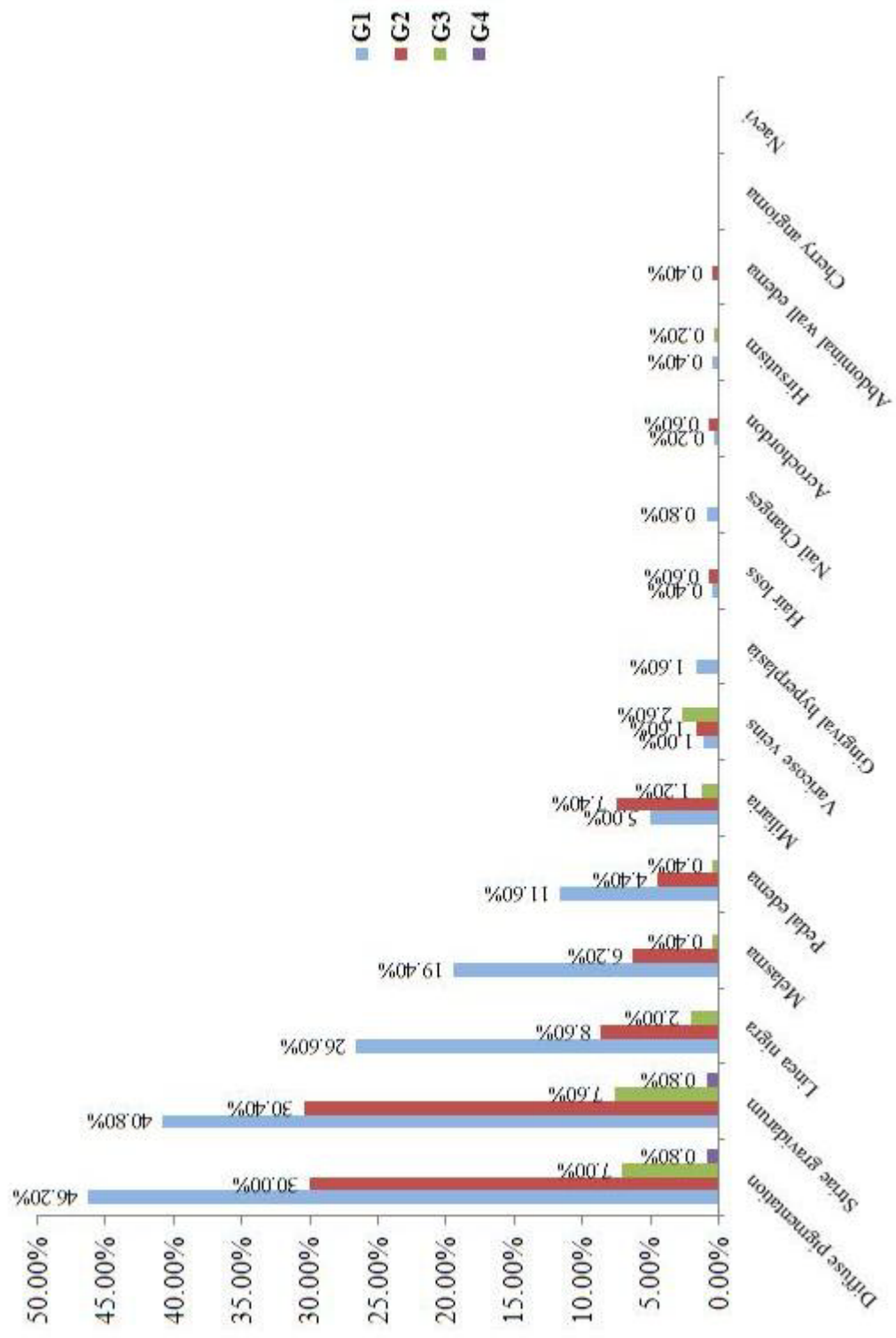


Fig 2c. Distribution of physiological changes among various trimesters

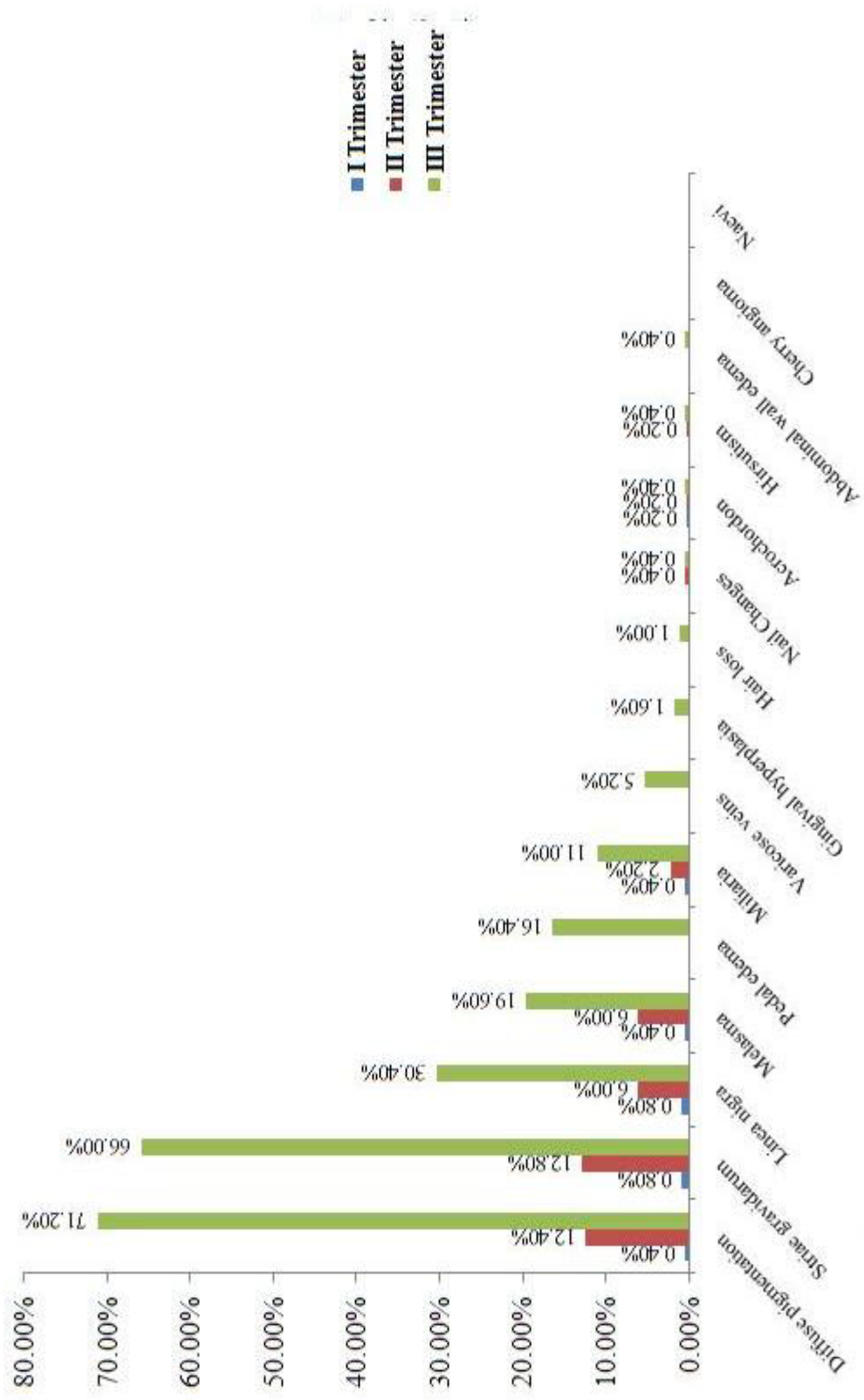


Table 3

Distribution of physiological changes observed during pregnancy(n=500)															
Physiological Changes	I Trimester				II Trimester				III Trimester				Total	Percentage (n=500)	
	G1	G2	G3	G4	G1	G2	G3	G4	G1	G2	G3	G4			
Pigmentation													454	90.80%	
Linea nigra	1	1	2	0	18	12	0	0	114	30	8	0	186	37.20%	
Melasma	0	2	0	0	22	8	0	0	75	21	2	0	130	26.00%	
Diffuse pigmentation	0	2	0	0	22	30	6	4	209	118	29	0	420	84.00%	
Naevi	0	0	0	0	0	0	0	0	0	0	0	0	0	0.00%	
Vascular													118	23.60%	
Gingival hyperplasia	0	0	0	0	0	0	0	0	8	0	0	0	8	1.60%	
Varicose veins	0	0	0	0	0	0	0	0	5	8	13	0	26	5.20%	
Pedal edema	0	0	0	0	0	0	0	0	58	22	2	0	82	16.40%	
Abdominal wall edema	0	0	0	0	0	0	0	0	0	2	0	0	2	0.40%	
Cherry angioma	0	0	0	0	0	0	0	0	0	0	0	0	0	0.00%	
Connective Tissue													402	80.40%	
Striae gravidarum	0	2	2	0	21	32	7	4	183	118	29	0	398	79.60%	
Acrochordon	0	1	0	0	0	1	0	0	1	1	0	0	4	0.80%	
Hair													8	1.60%	
Hirsutism	0	0	0	0	0	0	1	0	2	0	0	0	3	0.60%	
Hair loss	0	0	0	0	0	0	0	0	2	3	0	0	5	1.00%	
Eccrine													68	13.60%	
Miliaria	0	2	0	0	0	9	2	0	25	26	4	0	68	13.60%	
Nail													4	0.80%	
Nail Changes	0	0	0	0	2	0	0	0	2	0	0	0	4	0.80%	

Physiological skin changes were seen in about 474 cases (94.8%). Pigmentary changes were noticed in 454 cases (90.8%). Of the pigmentary changes, diffuse pigmentation was noted in 420 cases (84%). About 231 cases (46.2%) were primigravidas. About 356 cases (71.2%) were observed in III trimester. The pigmentation varied from brown to

black in colour. It was seen most commonly in the areolar region (100%) followed by genitalia, neck and axillary area. Secondary areolae was seen in 420 cases.

Site of maximum pigmentation among the pregnant women (n=420) is shown in Table 4.

Table 4

Site of pigmentation	Number of Cases	Percentage
Areola	420	100
Genitalia	180	42.86
Axilla	18	4.28
Neck	23	5.47
Generalised	120	28.57

Linea nigra was noticed in 186 cases (37.2%). This was observed in 30.4% (n=152) in III trimester, and majority 26.6% (n=133) were primigravidas. This was seen as hyperpigmented midline streak extending from the pubis symphysis to the xiphoid process of the sternum (**Fig 3**).

Melasma was seen in 130 cases (26%). This was noticed in the III trimester in 98 cases (19.6%), and most of them (n=97, 19.4%) were primigravidas. The cheeks and nose were the most commonly affected sites (**Fig 4**). Other sites involved were the forehead and chin.



Fig 3. Linea nigra in a primigravida in III trimester

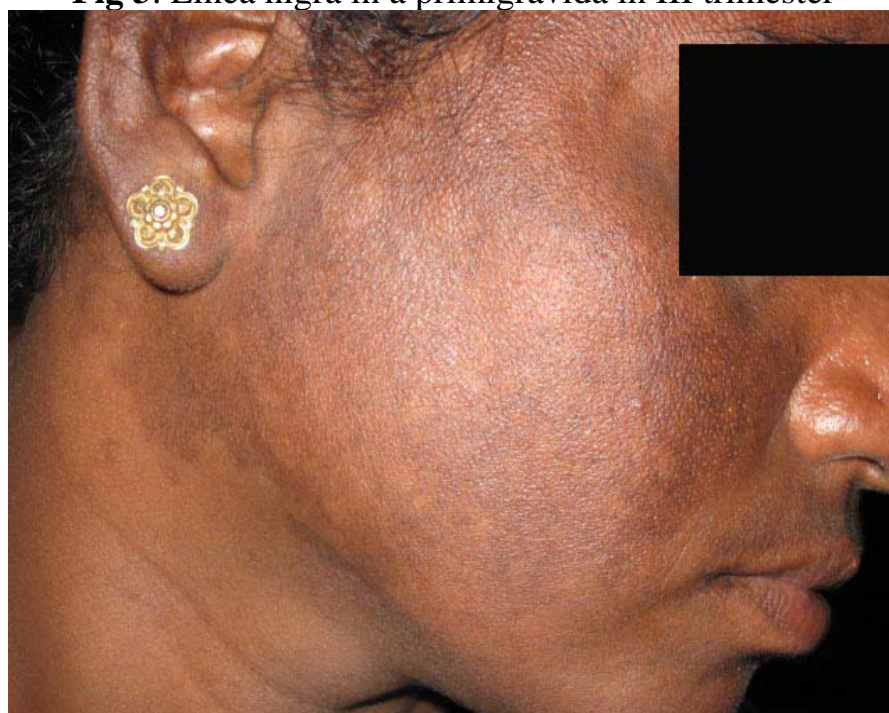


Fig 4. Melasma in a second gravida in III trimester

No increase in the number and size of melanocytic naevi was observed during the course of pregnancy in any case in this study.

Table 5
Statistical analysis of association between pigmentary changes and gravidity.

Pigmentary changes	Present	Absent	Test of significance
Primigravida	254 (50.8%)	37 (7.4%)	P = 0.001
Multigravida	200 (40%)	9(1.8%)	

50.8% of primigravida and 40% of multigravida had pigmentary changes, this difference in proportion of pigmentary changes between primigravida and multigravida was statistically significant (p=0.001) by Fisher's exact test.

Table 6
Statistical analysis of association between pigmentary changes and various trimesters.

Pigmentary changes		Present	Absent	Test of significance
Trimester	I	7 (1.4%)	2 (0.4%)	P = 0.349
	II	78 (15.6%)	9(1.8%)	
	III	369 (73.8%)	35 (7%)	

No statistically significant difference in proportion of pigmentary changes between various trimesters was observed in our study by Chi square test (p=0.349).

Vascular changes were seen in 118 cases (23.6%). Gingival hyperplasia was seen in 8 cases (1.6%), all were primigravidas seen in III trimester (**Fig 5**). Vulval varicosities were observed in 26 cases (5.2%). All were seen in III trimester. Majority of them were third gravida (n=13, 2.6%) (**Fig 6**). Nonpitting pedal edema was noticed in 82 cases (16.4%). Most of them were primigravidas (n=58, 11.6%). All were seen in III trimester (**Fig 7**). Abdominal wall edema was noticed in 2 cases (0.4%), both of them were second gravida seen in III trimester (**Fig 8**).

Table 7

Statistical analysis of association between vascular changes and gravidity.

Vascular changes	Present	Absent	Test of significance
Primigravida	73(14.6%)	218 (43.6%)	P = 0.3935
Multigravida	45 (9%)	164 (32.8%)	

By Fisher's exact test, the difference in proportion of vascular changes between primigravida and multigravida was not statistically significant (p=0.3935).



Fig 5. Gingival hyperplasia in a primigravida in III trimester



Fig 6. Vulval varicosity in a third gravida in III trimester



Fig 7. Nonpitting pedal edema in a primigravida in III trimester



Fig 8. Abdominal wall edema in a second gravida in III trimester



Fig 9. Striae gravidarum in a primigravida in III trimester

Table 8

Statistical analysis of association between vascular changes and various trimesters.

Pigmentary changes		Present	Absent	Test of significance
Trimester	I	0	9 (1.8%)	P < 0.0001
	II	0	87(17.4%)	
	III	118 (23.6%)	286 (57.2%)	

The difference in proportion of vascular changes between various trimesters was statistically significant ($p < 0.0001$) by Chi square test.

Connective tissue changes were observed in 402 cases (80.4%). Of these, striae gravidarum was seen in 398 cases (79.6%). 138 cases had striae due to previous pregnancies. 66% (n=330) were in III trimester. 40.8% (n=204) were primigravidas and 38.8% (n=194) were multigravidas. Among the multigravidas, 152 cases (30.4%) were second gravida , 38 cases (7.6%) were third gravida and 4 cases (0.8%) were fourth gravid. The abdomen was the most common site involved (**Fig 9**).

Table 9
Statistical analysis of association between striae gravidarum and gravidity.

Striae gravidarum	Present	Absent	Test of significance
Primigravida	204 (40.8%)	87 (17.4%)	P < 0.0001
Multigravida	194 (38.8%)	15 (3%)	

The difference in proportion of striae gravidarum between primigravida and multigravida was statistically significant ($p < 0.0001$) by Fisher's exact test.

Table 10
Statistical analysis of association between striae gravidarum and various trimesters.

Striae gravidarum		Present	Absent	Test of significance
Trimester	I	4 (0.8%)	5 (1%)	P = 0.007
	II	64 (12.8%)	23 (4.6%)	
	III	330 (66%)	74 (14.8%)	

Between various trimesters, statistically significant difference ($p = 0.007$) in proportion of striae gravidarum was observed by Chi square test.

Acrochordons was observed in 4 cases (0.8%) and 2 cases (0.4%) were in III trimester (**Fig 10**). 3 cases (0.6%) were second gravida and 1 case (0.2%) – primigravida.

Hirsutism was seen in 3 cases (0.6%). 2 cases (0.4%) were primigravidas seen in III trimester. Diffuse hair loss was seen in 5 cases (1%). All were in III trimester.

Nail changes were noted in 4 cases (0.8%). They included brittleness and leukonychia.

Miliaria was observed in 68 cases (13.6%). The lesions were distributed over the trunk, neck and face (**Fig 11**). 55 cases (11%) were in III trimester. Gravida 1 constituted 25 cases (5%), gravida 2 – 37 cases (7.4%), gravida 3 – 6 cases (1.2%).

The specific dermatoses of pregnancy were observed in 70 cases (14%) exclusively in III trimester in this study, the distribution of cases were shown in Table 11 (**Fig 12a**).

Table 11

Distribution of specific dermatoses observed during pregnancy(n=500)														
Dermatoses	I Trimester				II Trimester				III Trimester				Total	Percentage(n=500)
	G1	G2	G3	G4	G1	G2	G3	G4	G1	G2	G3	G4		
Pruritus gravidarum	0	0	0	0	0	0	0	0	44	8	0	0	52	10.40%
PUPPP	0	0	0	0	0	0	0	0	7	1	0	0	8	1.60%
Prurigo of pregnancy	0	0	0	0	0	0	0	0	6	3	0	0	9	1.80%
Pruritic folliculitis	0	0	0	0	0	0	0	0	1	0	0	0	1	0.20%
Pemphigoid gestationis	0	0	0	0	0	0	0	0	0	0	0	0	0	0.00%



Fig 10. Acrochordons and acanthosis nigricans in a second gravida in III trimester



Fig 11. Miliaria rubra in a second gravida in III trimester

Fig 12a. Distribution of specific dermatoses observed during pregnancy(n=500)

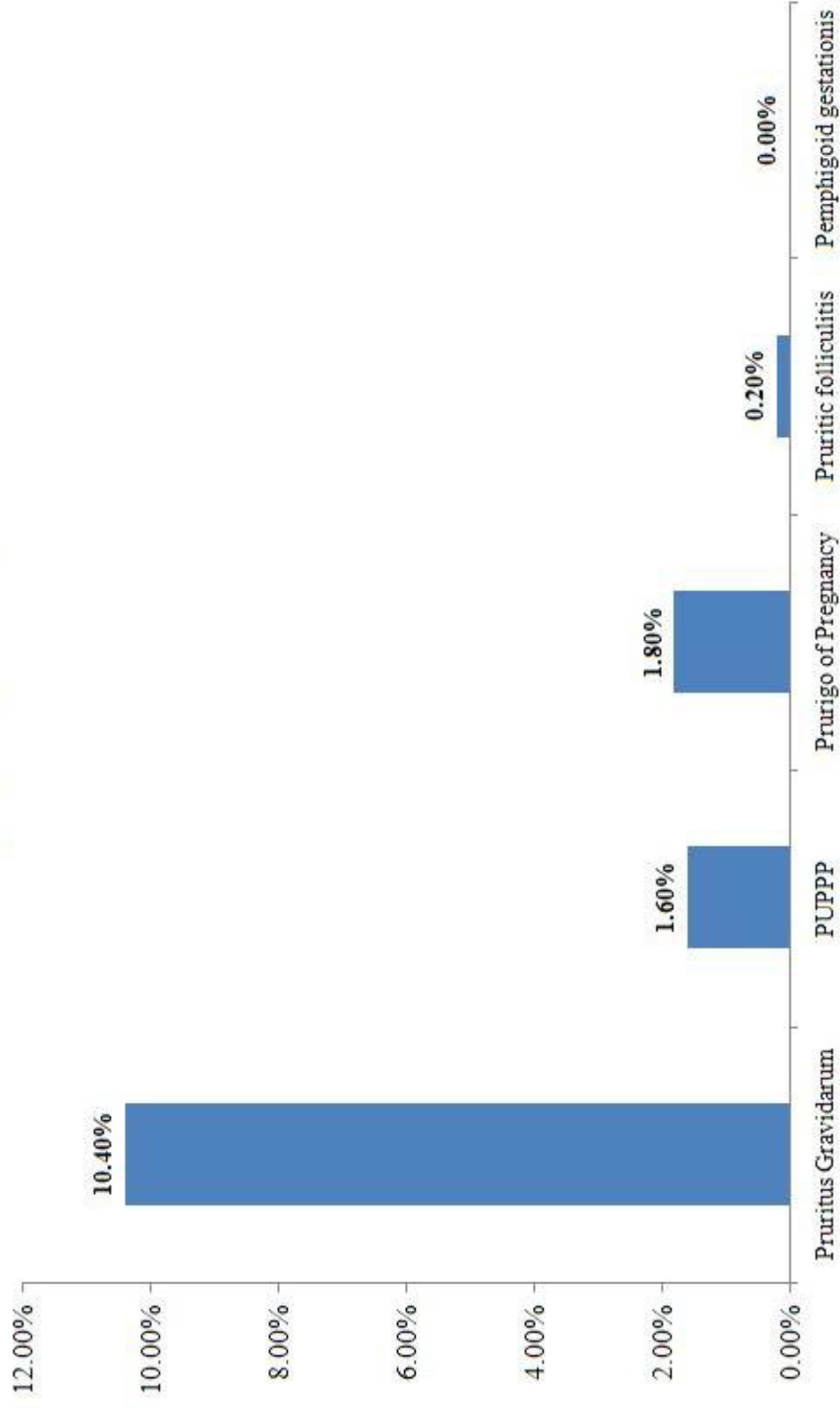


Fig 12b. Distribution of specific dermatoses among various gravidas

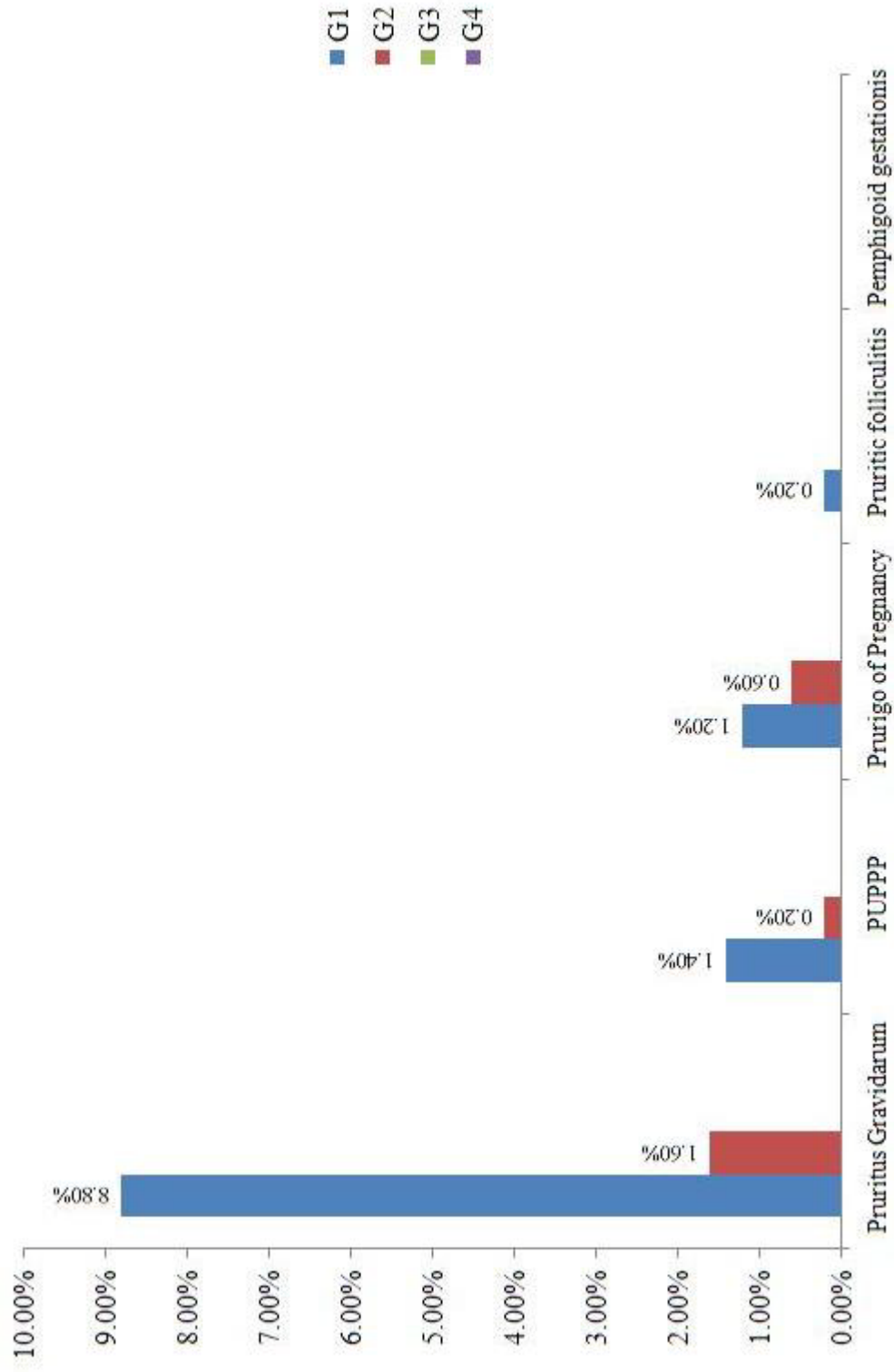
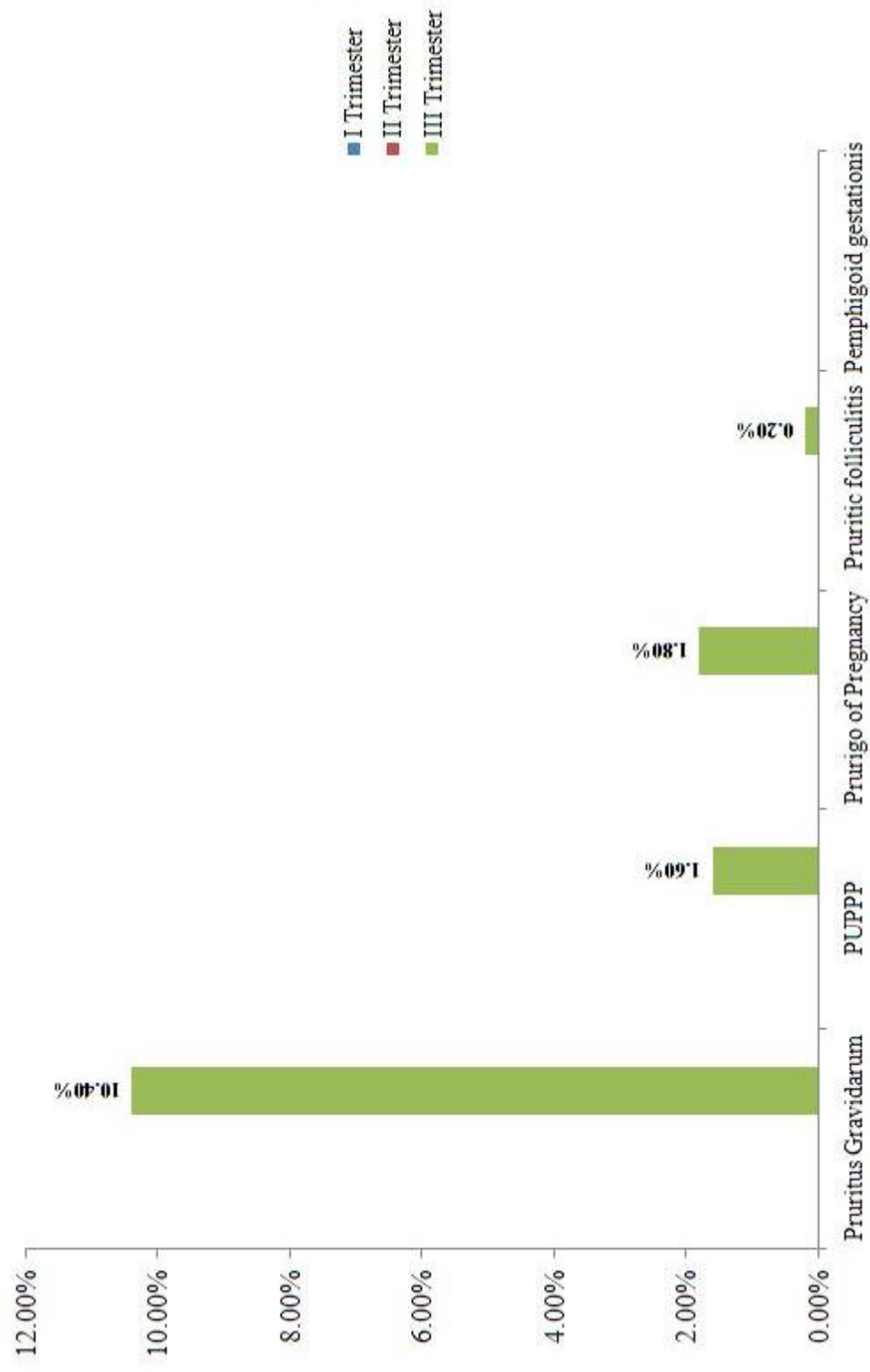


Fig 12c. Distribution of specific dermatoses among various trimesters



The distribution of specific dermatoses of pregnancy among various gravidas is shown in **Fig 12b**.

The distribution of specific dermatoses of pregnancy among various trimesters is shown in **Fig 12c**.

Table 12
Statistical analysis of association between specific dermatoses and gravidity.

Specific dermatoses	Present	Absent	Test of significance
Primigravida	58(11.6%)	233(46.6%)	P < 0.0001
Multigravida	12 (2.4%)	197 (39.4%)	

By Fisher's exact test, the difference in proportion of specific dermatoses between primigravida and multigravida was statistically significant ($p < 0.0001$).

Table 13
Statistical analysis of association between specific dermatoses and various trimesters.

Specific dermatoses		Present	Absent	Test of significance
Trimester	I	0	9 (1.8%)	P < 0.0001
	II	0	87 (17.4%)	
	III	70 (14%)	334 (66.8%)	

By Chi square test, the difference in proportion of specific dermatoses between various trimesters was statistically significant ($p < 0.0001$).

Pruritus gravidarum was the most common disease encountered ($n=52$, 10.4%). Of those affected, 44 (8.8%) were primigravidas and 8 were gravida 2 (1.6%). All had single gestation pregnancies. Two of the 8 multigravidas reported identical symptoms in previous pregnancies. All the cases presented exclusively in III trimester. All had sudden onset of severe pruritus which started initially in the abdomen and later became generalized, which was followed by secondary skin lesions, namely excoriations and excoriated papules. None of these cases had clinical jaundice. The liver function tests were normal in all except two patients who had elevated alkaline phosphatase levels.

Pruritic urticarial papules and plaques of pregnancy were observed in 8 cases (1.6%). Majority were primigravidas ($n=7$, 1.4%) and 1(0.2%) was a multigravida (G2). All had single gestation pregnancy. Skin lesions occurred in all cases in III trimester. The pruritic eruption started characteristically on the lower abdomen, particularly within or adjacent to the striae distensae in all cases (**Fig 13a, 13b&13c**). With disease progression, the eruption was seen to involve other parts of the body also (**Fig 14&15**). Vast majority of patients had erythematous urticarial papules and plaques.



Fig 13a

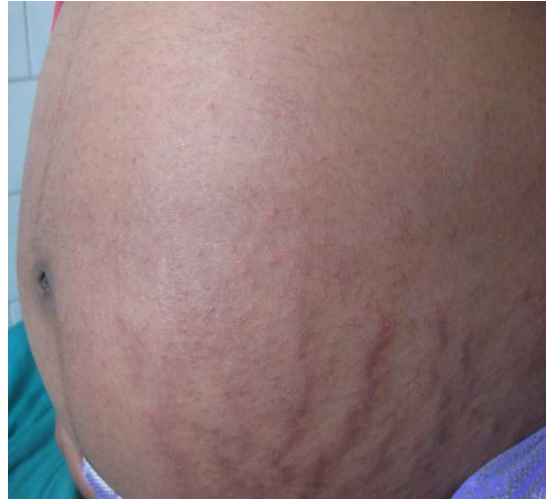


Fig 13b



Fig 13c

Urticarial papules over striae in primigravida with PUPPP in
III trimester (**Fig 13a, Fig 13b&13c**)



Fig 14. Urticarial papules over legs in a primigravida with PUPPP in III trimester



Fig 15. Urticarial papules and plaques over thighs in a primigravida with PUPPP in III trimester

Prurigo of pregnancy was seen in 9 cases (1.8%). 6 (1.2%) were primigravidas and 3 (0.6%) were multigravidas. All had onset of skin lesions in III trimester. All patients had single gestation pregnancies none of the multiparous women reported similar rashes in previous pregnancies. The eruption was typically composed of itchy hyperpigmented papules and excoriations which first appeared over extensor surfaces of limbs (**Fig 16**), and with disease progression, involved the abdomen and trunk (**Fig 17&18**).

Pruritic folliculitis was observed in 1 case (0.2%), which was seen in a primigravida in III trimester, as itchy erythematous follicular papules over the abdomen (**Fig 19**).

The face and mucous membrane were spared in all the specific dermatoses. No case of pemphigoid gestationis was observed in this study.

Infections were observed in 30.8% (n=154) of cases, which is shown in Table 14 (**Fig 20a**).

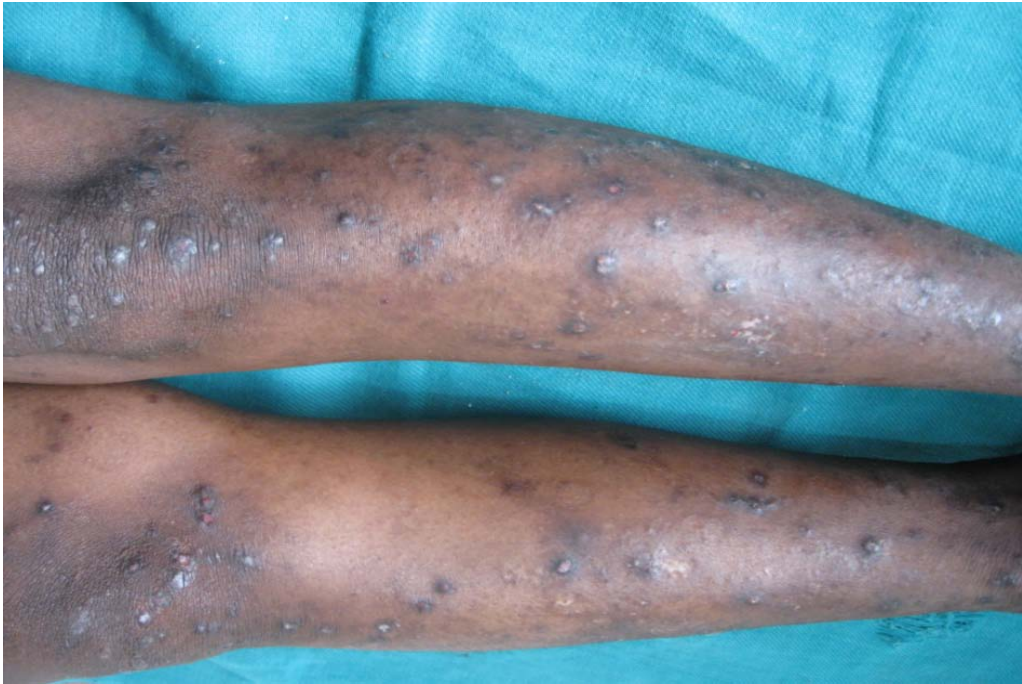


Fig 16. Excoriated papules and nodules over extensor surface of legs in a primigravida with prurigo of pregnancy in III trimester



Fig 17. Excoriated papules and nodules over abdomen in a primigravida with prurigo of pregnancy in III trimester



Fig 18. Excoriated papules and nodules over forearms in a primigravida with prurigo of pregnancy in III trimester



Fig 19. Follicular papule over abdomen in a primigravida with pruritic folliculitis in III trimester

Table 14

Distribution of infections observed during pregnancy(n=500)														
Infections	I Trimester				II Trimester				III Trimester				Total	Percentage (n=500)
	G1	G2	G3	G4	G1	G2	G3	G4	G1	G2	G3	G4		
Bacterial														
Furunculosis	0	0	0	0	0	0	0	0	4	0	0	0	4	0.80%
Hansen's Disease	1	0	0	0	0	0	0	0	1	0	0	0	2	0.40%
Viral														
Wart	0	0	0	0	1	0	0	0	6	1	0	0	8	1.60%
HSV	0	1	1	0	0	0	0	0	1	2	0	0	5	1.00%
Herpes zoster	0	0	0	0	0	0	0	0	2	4	0	0	6	1.20%
HIV	0	0	0	0	1	0	0	0	0	0	0	0	1	0.20%
Fungal														
Candidiasis	0	0	0	0	1	3	1	0	13	18	4	0	40	8.00%
Tinea versicolor	0	0	0	0	13	4	1	0	19	12	4	0	53	10.60%
Dermatophyte	0	0	0	0	3	2	0	2	9	8	2	0	26	5.20%
Protozoal														
Trichomoniasis	0	0	0	0	0	3	0	0	2	3	0	0	8	1.60%
Arthropod														
Scabies	0	0	0	0	0	1	0	0	0	0	0	0	1	0.20%

The distribution of infections among various gravidas is shown in **Fig 20b.**

The distribution of infections among various trimesters is shown in **Fig 20c.**

Fig 20a. Distribution of infections observed during pregnancy

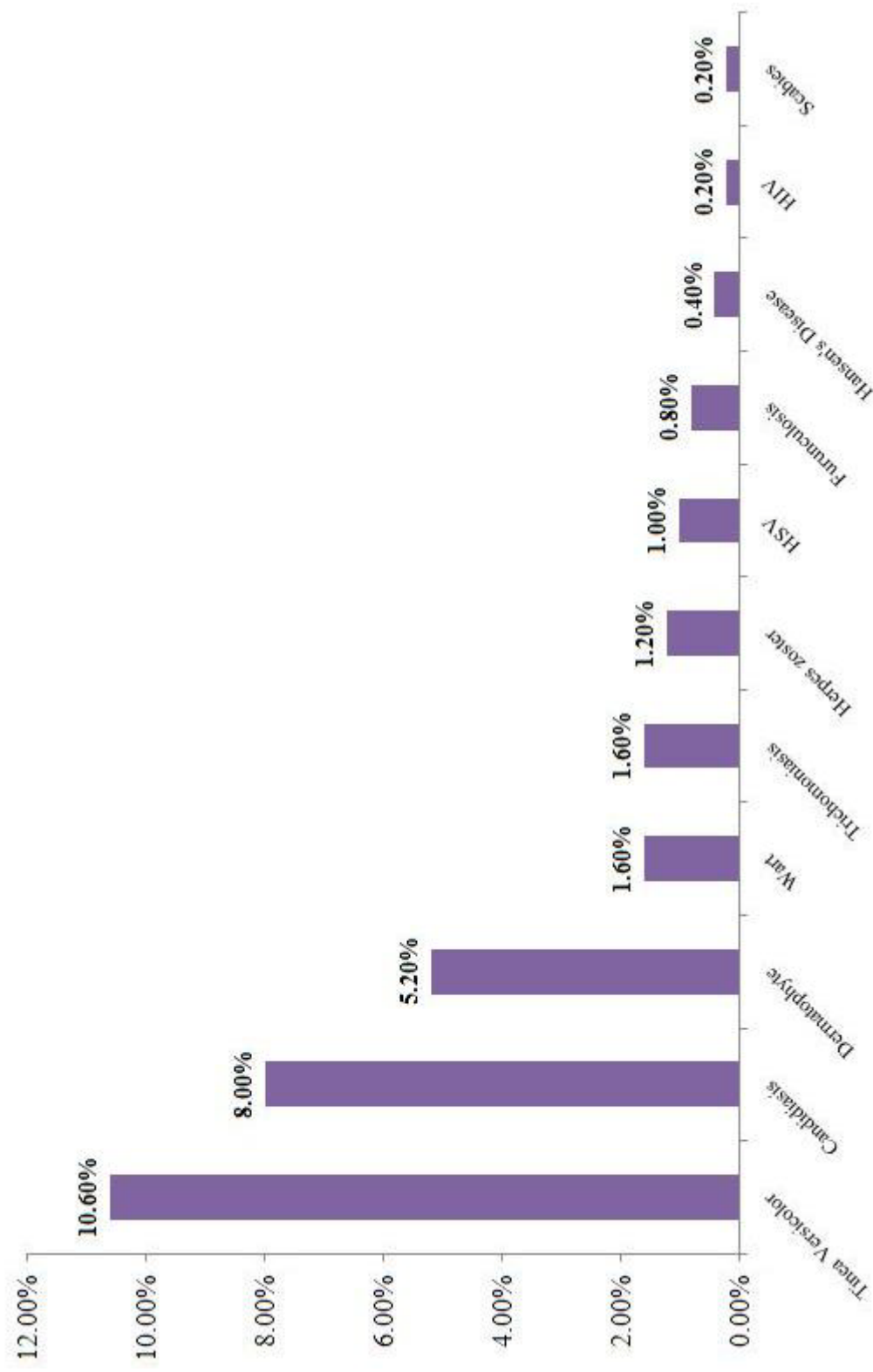


Fig 20b. Distribution of infections among various gravidas

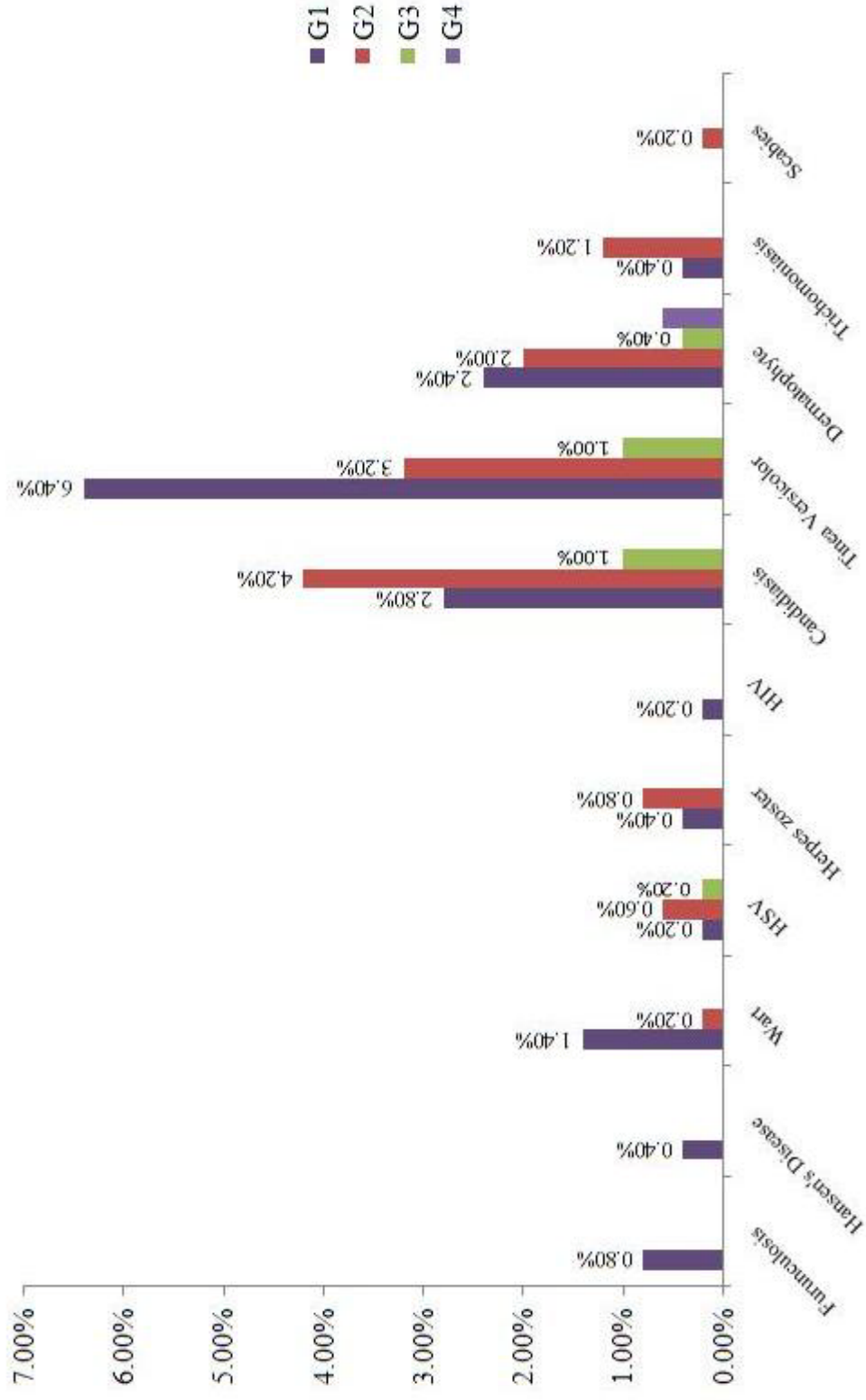


Fig 20c. Distribution of infections among various trimesters

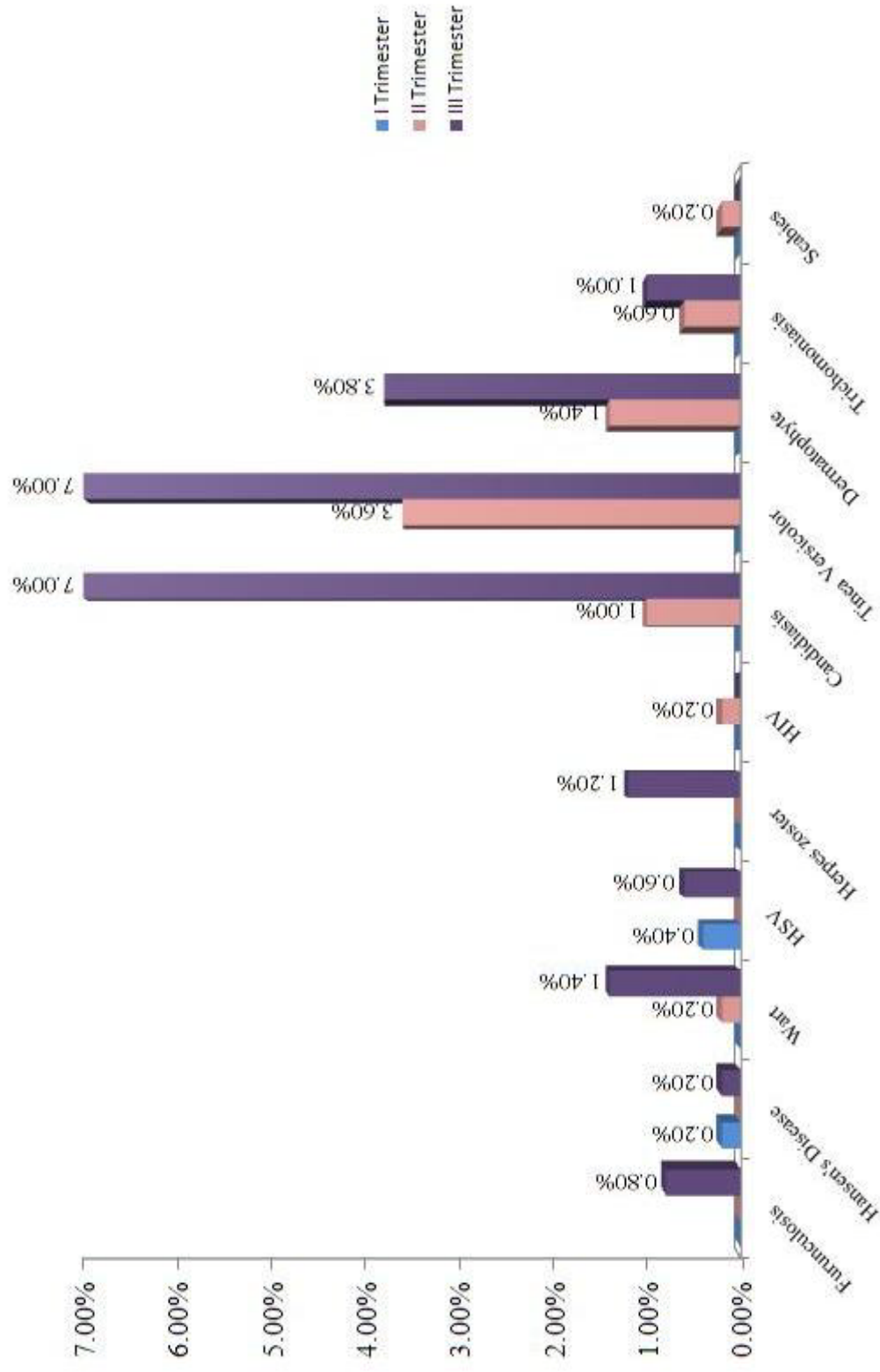


Table 15
Statistical analysis of association between infections and gravidity.

Infections	Present	Absent	Test of significance
Primigravida	77 (15.4%)	214 (42.8%)	P = 0.0142
Multigravida	77 (15.4%)	132 (26.4%)	

Fisher's exact test showed statistically significant difference in proportion of infections between primigravida and multigravida (p=0.0142).

Table 16
Statistical analysis of association between infections and various trimesters.

Infections		Present	Absent	Test of significance
Trimester	I	3 (0.6%)	6 (1.2%)	P = 0.059
	II	36 (7.2%)	51 (10.2%)	
	III	115 (23%)	289 (57.8%)	

Chi square test showed no statistically significant difference in proportion of infections between various trimesters (p=0.059).

Bacterial infections were observed in 1.2% (n=6). Of the bacterial infections, furunculosis was observed in 4 primigravidas (0.8%). They were in III trimester (**Fig 21**). Out of these 4 cases, one had gestational diabetes.

Hansen disease was seen in 2 cases (0.4%). One was a primigravida in III trimester who presented as lepromatous leprosy with healed trophic ulcer (**Fig 22a&22b**). Other was a primigravida in I trimester who presented as borderline tuberculoid leprosy.

Viral infections were observed in 4% (n=20). Of the viral infections, wart was seen in 8 cases (1.6%). Condyloma acuminata was noted in 5 cases (1%), all were primigravidas in III trimester. One of them had verrucous pinkish exuberant growth occupying the introitus (**Fig 23**). The other women had pinkish and verrucous papules over the labia minora and labia majora. 3 cases (0.6%) of verruca vulgaris was noticed. Of these, 2 cases were primigravidas and 1 was a second gravida (**Fig 24**).

Herpes simplex infection was noted in 5 cases (1%). Of these, herpes labialis (HSV 1) was seen in 3 cases (0.6%). They presented with multiple grouped vesicles and erosions around the mouth (**Fig 25**). HSV II (genital herpes) was seen in 2 cases (0.4%), who presented with multiple painful erosions and ulcers over the labia majora and minora (**Fig 26**). Tzanck smear showed multinucleated giant cells. Herpes zoster was observed in 1.2% (n=6) of cases (**Fig 27a&27b**).



Fig 21. Furunculosis over face and neck in a primigravida in III trimester



Fig 22a



Fig 22b

Fig 22a&22b. Infiltration over forehead, cheeks and chin in a primigravida with lepromatous leprosy in III trimester



Fig 23. Condyloma acuminata in a primigravida in III trimester



Fig 24. Verruca vulgaris in a primigravida in III trimester



Fig 25. Herpes labialis in a primigravida in III trimester

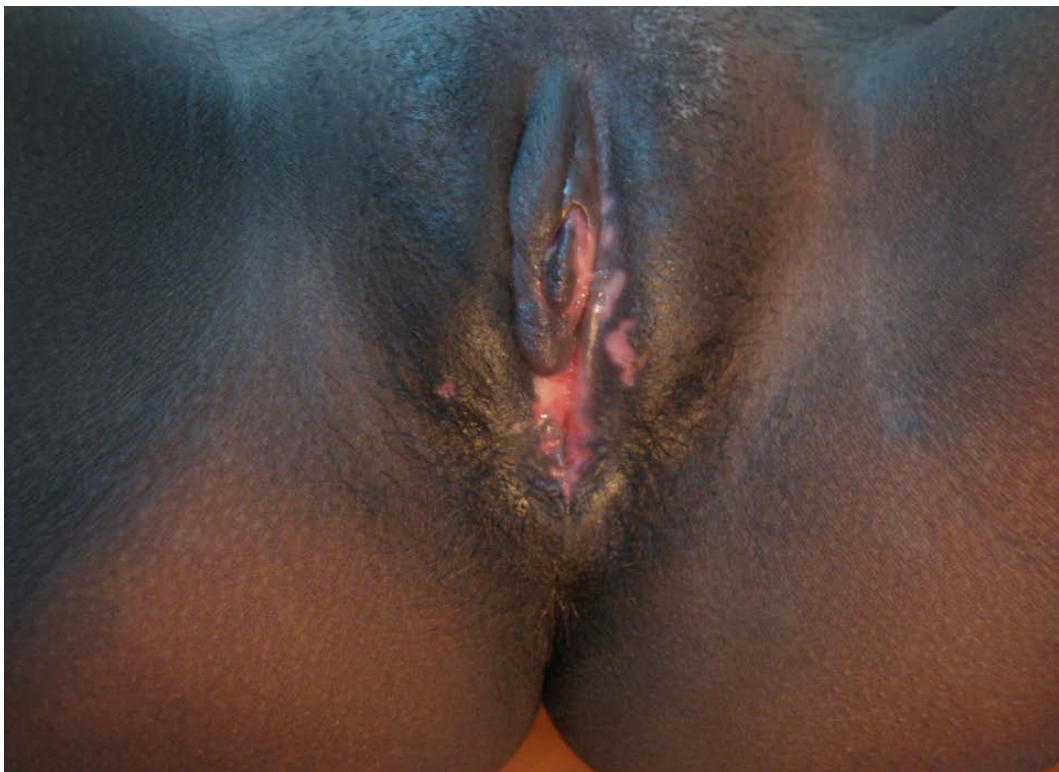


Fig 26. Genital herpes over labia minora and majora in a second gravida in I trimester



Fig 27a



Fig 27b

Fig 27a&27b. Herpes zoster involving T4 dermatome in a primigravida in III trimester

HIV was noticed in 1 case (0.2%) She was a primigravida seen in II trimester. Male partner was also infected with HIV. She had exaggerated insect bite allergy.

Fungal infections were observed in 23.8% (n=119). Of the fungal infections, pityriasis versicolor was the commonest (n = 53, 10.6%). It was seen in 32 cases (6.4%) of primigravidas, 16 cases (3.2%) second gravida and 5 cases (1%) of third gravida. Most of them were in III trimester (n=35, 7%). It was seen as hypopigmented fine scaly macules and patches distributed over face, chest, and upper back, few were perifollicular (**Fig 28**). Scraping and 10% potassium hydroxide mount showed short, angulated, hyaline, nonseptate hyphae and blastospores.

Candidiasis was observed in 40 cases (8%). Most of them were in third trimester (n=35, 7%). Candidal vulvovaginitis and intertrigo (**Fig 29a**) was observed in 34 cases (6.8%). Oral candidiasis (**Fig 29b**) was seen in 6 cases (1.2%). Scraping and 10% potassium hydroxide mount showed pseudohyphae with blastospores. 13 of them had associated gestational diabetes.

Dermatophytosis was observed in 26 cases (5.2%). Most of them were in III trimester (n = 19, 3.8%). 12 were primigravidas (2.4%), 10 were second gravida (2%), 2 were third gravida (0.4%) and 2 were fourth



Fig 28. Pityriasis versicolor in a primigravida in II trimester



Fig 29a. Candidal vulvovaginitis and intertrigo in a second gravida in III trimester



Fig 29b. Oral candidiasis with angular cheilitis in a primigravida in III trimester



Fig 30. Tinea corporis in a second gravida in III trimester

gravida (0.4%). The clinical types seen were tinea corporis, tinea cruris, tinea axillaris and tinea glutealis. The lesions were annular with erythematous, scaly borders and few papules in the margins with central clearance (**Fig 30**). Scraping and 10% potassium hydroxide mount showed branching, septate, hyaline hyphae with arthrospores.

Of the protozoal diseases, Trichomoniasis occurred in 8 cases (1.6%). It was observed to be more common in the III trimester (n = 5, 1%) and in second gravida (n=6, 1.2%), who presented with pruritic vaginal discharge. Wet film showed *Trichomonas vaginalis* organisms with characteristic motility.

Scabies was seen in 1 case (0.2%) in a second gravida in II trimester. She had itchy papules over the web spaces of the hands, thighs and genitalia.

The distribution of miscellaneous dermatoses observed in pregnancy is shown in Table 17 (**Fig 31a**).

The distribution of miscellaneous dermatoses observed in pregnancy among various gravidas is shown in **Fig 31b**.

The distribution of miscellaneous dermatoses observed in pregnancy among various trimesters is shown in **Fig 31c**.

Fig 31a. Miscellaneous dermatoses observed during pregnancy(n=500)

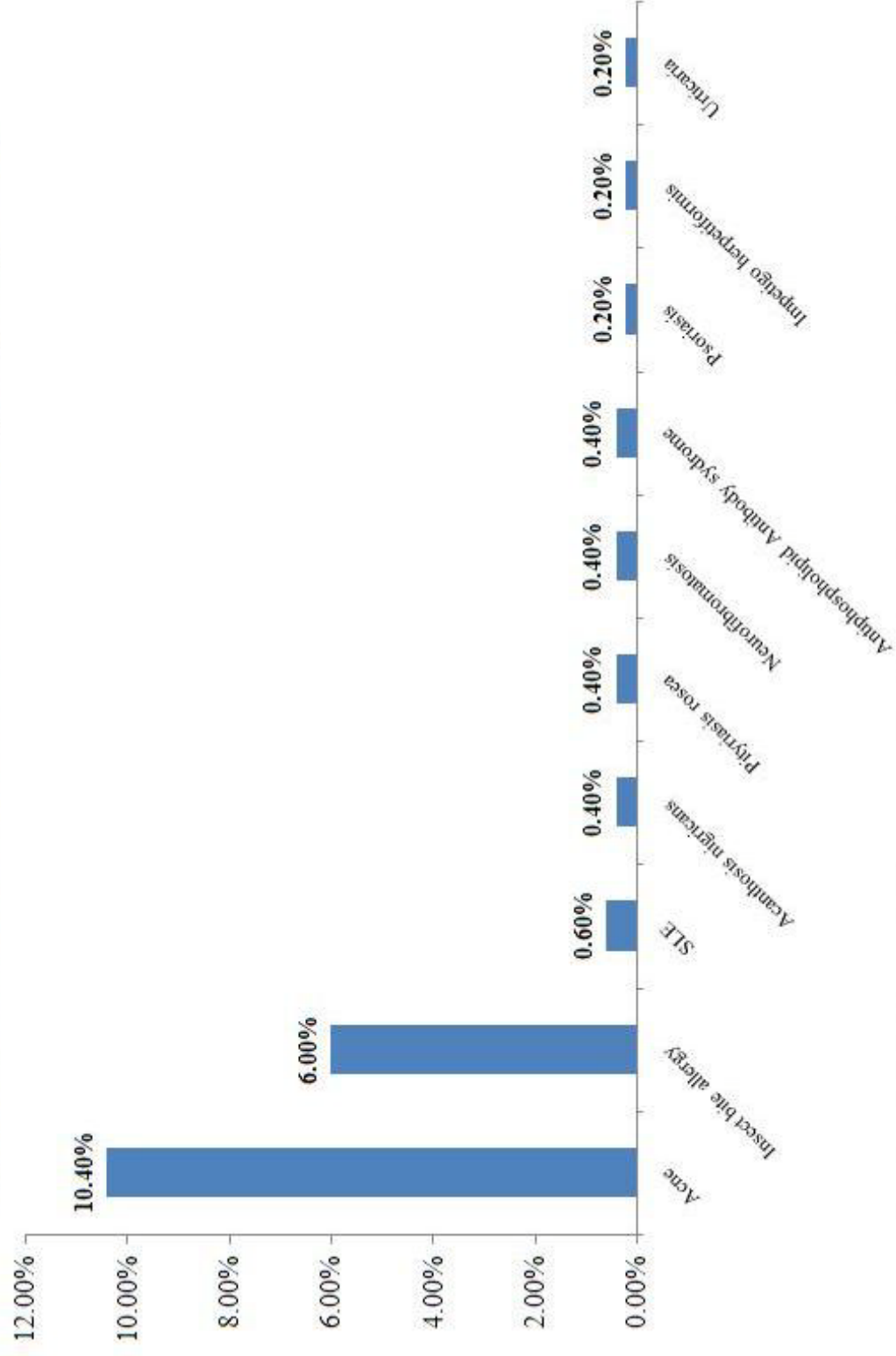


Fig 31b. Distribution of miscellaneous dermatoses among various gravidas

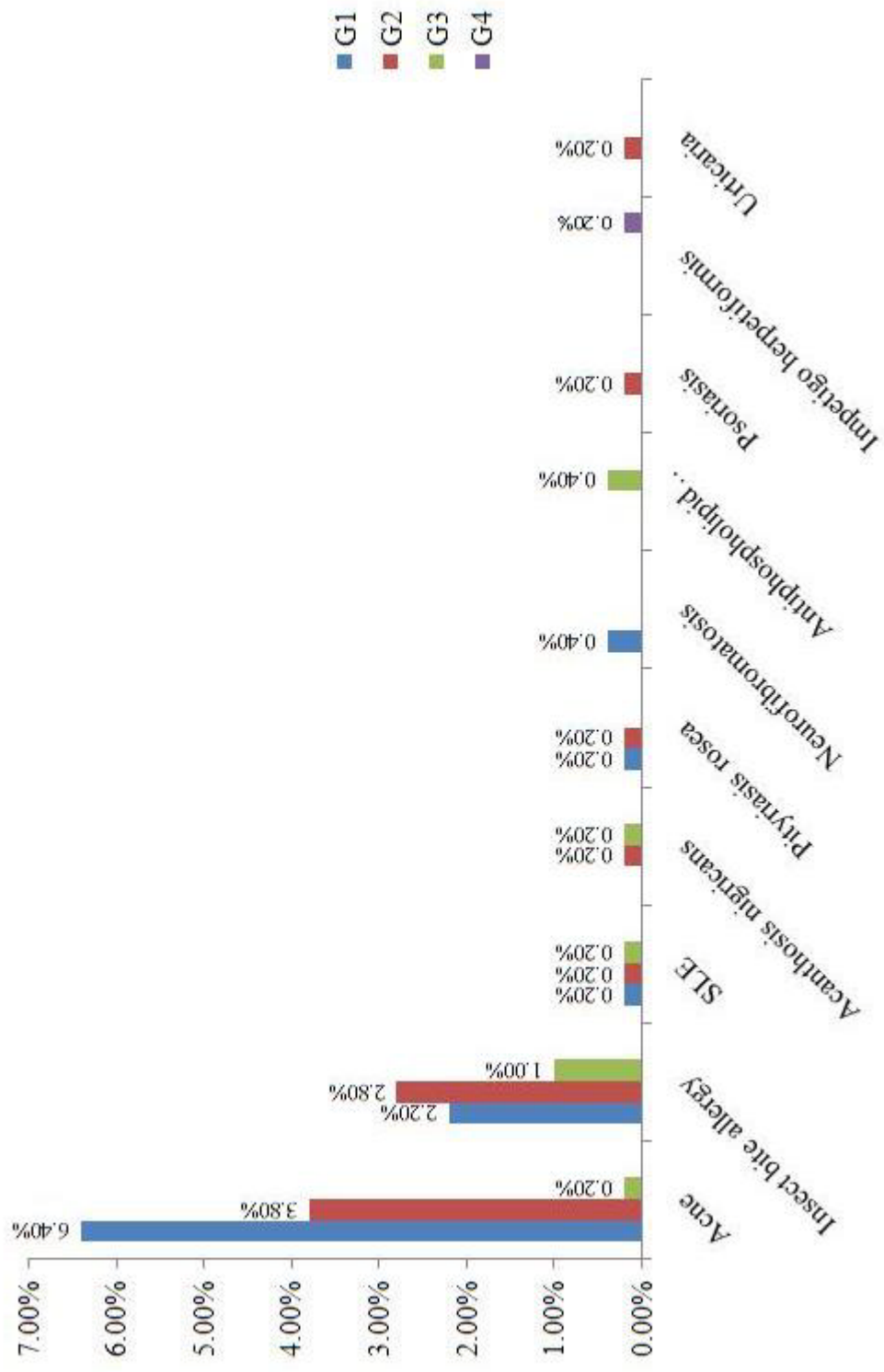


Fig 31c. Distribution of miscellaneous dermatoses among various trimesters

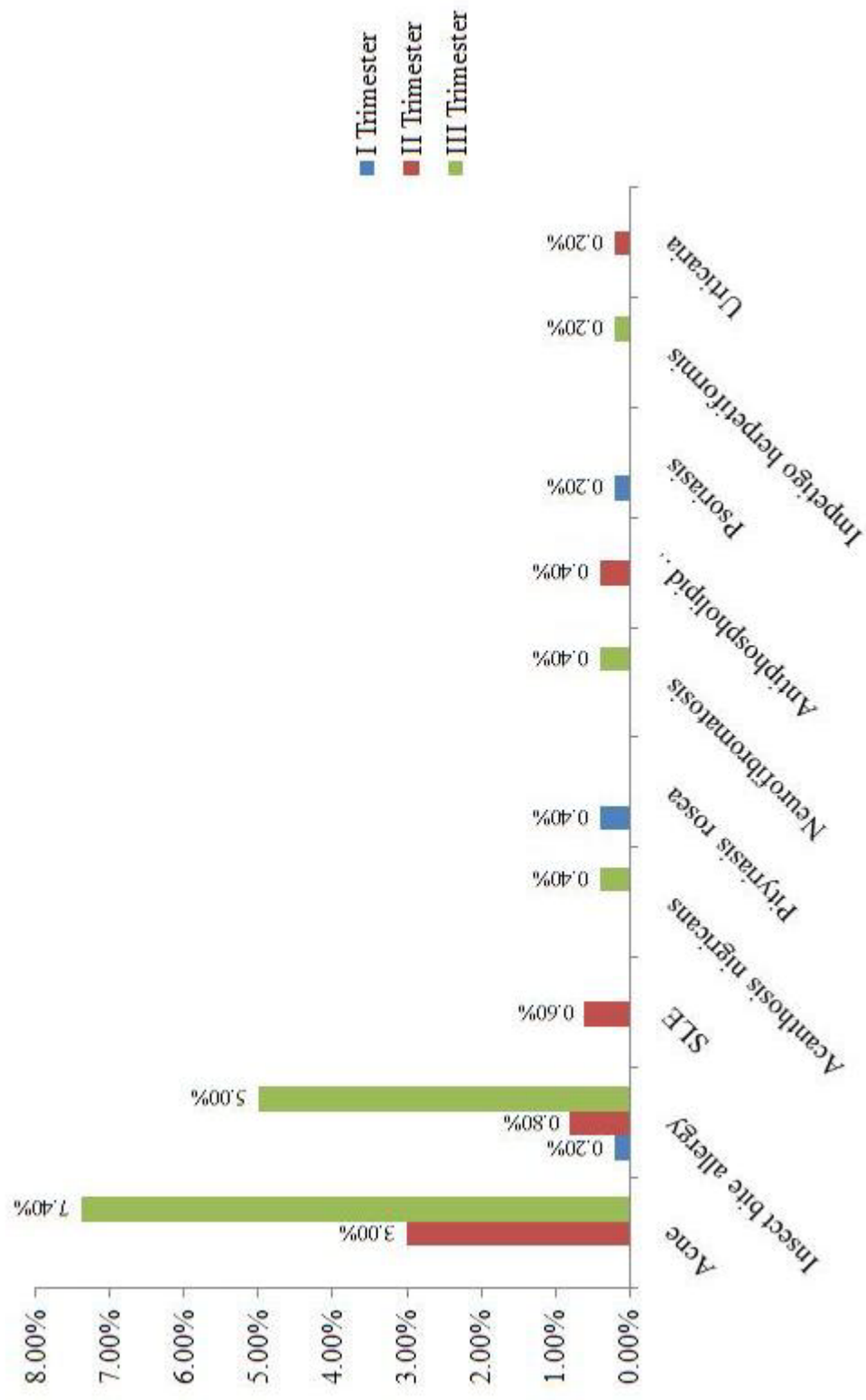


Table 17

Distribution of miscellaneous dermatoses observed during pregnancy(n=500)															
Dermatoses	I Trimester				II Trimester				III Trimester				Total	Percentage (n=500)	
	G1	G2	G3	G4	G1	G2	G3	G4	G1	G2	G3	G4			
Acne	0	0	0	0	9	6	0	0	23	13	1	0	52	10.40%	
Insect bite allergy	0	0	1	0	2	2	0	0	9	12	4	0	30	6.00%	
SLE	0	0	0	0	1	1	1	0	0	0	0	0	3	0.60%	
Acanthosis nigricans	0	0	0	0	0	0	0	0	0	1	1	0	2	0.40%	
Pityriasis rosea	1	1	0	0	0	0	0	0	0	0	0	0	2	0.40%	
Neurofibromatosis	0	0	0	0	0	0	0	0	2	0	0	0	2	0.40%	
Antiphospholipid antibody syndrome	0	0	0	0	0	0	2	0	0	0	0	0	2	0.40%	
Psoriasis	0	1	0	0	0	0	0	0	0	0	0	0	1	0.20%	
Impetigo herpetiformis	0	0	0	0	0	0	0	0	0	0	0	1	1	0.20%	
Urticaria	0	0	0	0	0	1	0	0	0	0	0	0	1	0.20%	

Acne vulgaris was observed in 52 cases (10.4%). Most of them were primigravidas (n = 32, 6.4%), 19 (3.8%) were second gravida and 1 (0.2%) was a third gravida. Most of them were seen in third trimester (n=37, 7.4%). 32 cases (6.4%) had onset of acne during pregnancy and 20 cases (4%) had exacerbation of previous acne lesions (**Fig 32**).

Insect bite allergy was observed in 30 cases (6%), most commonly seen in III trimester (n=25, 5%). 11 cases (2.2%) were primigravidas, 14 cases (2.8%) were second gravida, and 5 cases (1%) were third gravida. The lesions were itchy hyperpigmented papules and excoriations distributed mainly below the elbows and the knees.

Acanthosis nigricans was observed in 2 cases (0.4%) in III trimester. One was a second gravida (n = 1, 0.2%) (**Fig10**) and other a third gravida (n = 1, 0.2%), both of them were obese, presented with hyperpigmented velvety plaques over the neck.

Pityriasis rosea was observed in 2 cases (0.4%) in I trimester. One was a primigravida and other a second gravida, who presented with multiple erythematous patches and plaques with collarette of fine scales distributed over back, chest and abdomen (**Fig 33**).

Psoriasis was seen in 1 case (0.2%) in I trimester in a second gravida. She presented as chronic plaque type with well defined plaques covered with silvery scales over the extensor surface of arm, forearm, thighs, legs, abdomen and back. Auspitz sign was positive (**Fig 34**).

Impetigo herpetiformis (n = 1, 0.2%) occurred in a fourth gravida in III trimester. The patient was a known case of psoriasis vulgaris who presented as generalized pustular psoriasis during pregnancy, with lakes of pus over abdomen, thighs, legs, arms, forearms, back and intertriginous areas (**Fig 35**).

Urticaria was seen in 1 case (0.2%), the urticarial wheals developed in II trimester over the extremities, abdomen and chest.



Fig 32. Acne vulgaris in a primigravida in III trimester



Fig 33. Pityriasis rosea over back in a primigravida in I trimester

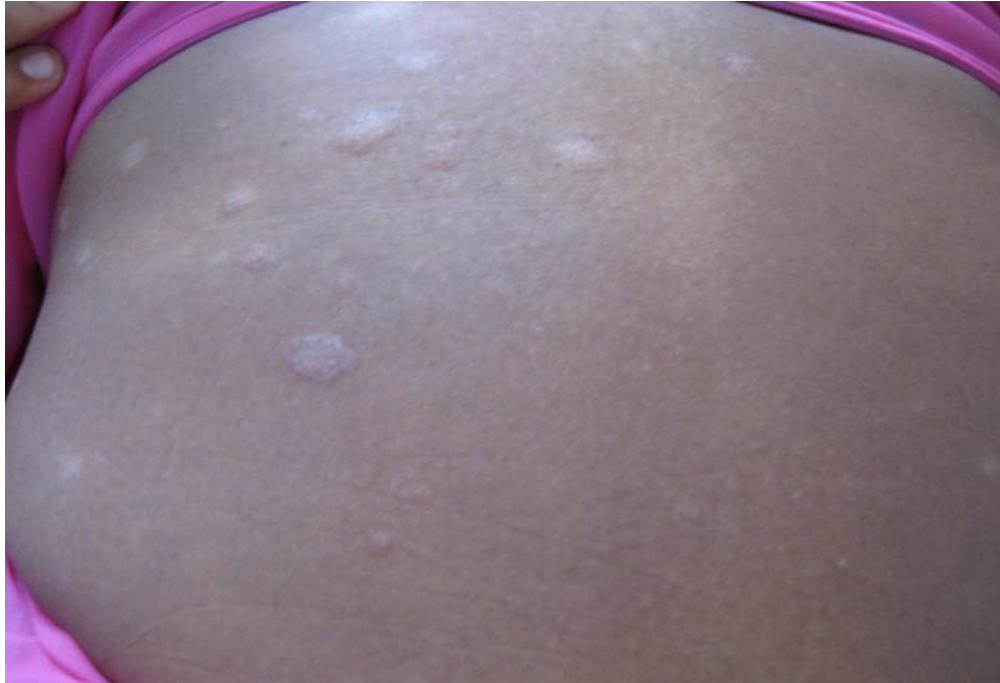


Fig 34. Psoriasis vulgaris in a second gravida in I trimester



Fig 35. Lakes of pus and pustules over abdomen in a fourth gravida with impetigo herpetiformis in III trimester

Exacerbation of neurofibromatosis was seen in 2 cases (n = 2, 0.4%). Both were primigravidas, with increased number of cutaneous neurofibromas during pregnancy, particularly in III trimester (**Fig 36**).

Antiphospholipid antibody syndrome was observed in 2 cases (0.4%). Both of them were third gravida seen in II trimester. None of them had cutaneous features but had lupus anticoagulant antibodies. 3 known cases (0.6%) of systemic lupus erythematosus were seen in II trimester. 1- primigravida, 1- second gravida and 1- third gravida. All had cutaneous flares and arthritis (**Fig 37a&37b**).



Fig 36. Neurofibromatosis in a primigravida in III trimester



Fig 37a



Fig 37b

Fig 37a&37b. Maculopapular rash in a primigravida with systemic lupus erythematosus in II trimester

DISCUSSION

Pregnancy produces many cutaneous changes, some of which are specifically related to pregnancy (dermatoses of pregnancy), some are modifiable by pregnancy and others that are more common are named physiologic. These physiological skin changes, usually do not impair the health of the mother or the fetus. Nevertheless, some can be cosmetically significant and of importance to the dermatologist.

In our study, majority of the pregnant women had physiological skin changes (n = 474, 94.8%). Hyperpigmentation is common during pregnancy and may be seen in upto 90% of pregnant women.¹ Muzaffar et al.⁸² observed pigmentary changes in 90.7% of cases. Kumari et al.² reported hyperpigmentation in 91.4% of pregnant women. In this study we observed pigmentary changes in 90.8% which is consistent with above mentioned studies. The most common pigmentary change was diffuse pigmentation seen in 420 cases (84%). The sites of increased pigmentation observed in the order of frequency were areolae, genitalia, neck and axilla. Secondary areolae developed in all 420 cases (100%). Linea nigra was seen in 186 cases (37.2%).

Melasma has been reported to occur in upto 70% of pregnant women.^{5,8} Muzaffar et al.⁸² found melasma to be present in 46.4% of their cases and Raj et al.⁸³ observed melasma in 8.8% of cases, whereas in this study, melasma was seen in 26% (n = 130) of cases. Cheeks and nose were the common sites involved. Martin and Leal khouri⁸⁴ reported an onset of melasma during II trimester, whereas in this study, the onset of melasma in most (n = 98, 19.6%) of the cases were in early third trimester.

Type B pigmentary demarcation lines are known to appear for the first time during pregnancy², which was not observed in our study. This may be due to the fact that pigmentary changes are more discernible in fair skinned individuals.

Higher proportion of pigmentary changes was observed in primigravidas (50.8%), which was statically significant (p=0.001).

Muzaffar et al.⁸² reported vascular changes in 34.2% of cases, with non-pitting pedal edema in 48.5% of cases. In our study, vascular changes were noticed in 23.6% (n = 118) of cases, with non-pitting pedal edema in 16.4% of cases.

Varicosities were reported in 40% of patients.¹⁷ Raj et al.⁸³ reported varicosities in 5.3%. In this study, vulval varicosities were observed in 5.2% (n=26) of cases, with the onset being in III trimester.

During pregnancy, gingival hyperplasia was seen in 80%, with the onset in III trimester.^{18, 19} In a study by Muzaffar et al.⁸² 16.4% of cases had gingival edema and redness. Kumari et al.² reported gingivitis in 1.5% of pregnant women. In this study, gingivitis not attributable to bad oral hygiene was seen in 1.6% (n = 8) of cases. Raj et al.⁸³ had seen 3 cases of pyogenic granuloma in their study. None of the pregnant women in our study had pyogenic granuloma.

In III trimester, higher proportion of vascular changes was observed (23.6%), which was statistically significant ($p<0.0001$).

Kumari et al.², Muzaffar et al.⁸², Raj et al.⁸³ and Shivakumar and Madhavamurthy⁸¹ reported striae gravidarum in 79.7%, 77.1%, 75.4% and 66.47% respectively. In our study, the prevalence of striae gravidarum was found to be 79.6% (n = 398) of cases, with the onset being more common during third trimester (n = 330, 66%), which was statistically significant ($p=0.007$). In primigravidas, higher proportion of striae gravidarum was observed (40.8%), which was statistically significant ($p<0.0001$).

Acrochordons was seen in upto 0.8% of individuals and are probably due to hormonal factors.³

The changes in hair growth such as hirsutism (0.6%), telogen effluvium (1%) are in accordance with other studies.²

Miliaria was observed in 13.6% (n = 68) of cases. This may be due to the increased eccrine function during pregnancy.^{8,14}

The nail changes observed were brittleness and leukonychia in 0.8% of cases, which is consistent with other studies.²

Specific dermatoses of pregnancy are almost always associated with pruritus and an eruption of variable severity. Kumari et al.² reported 22 (3.6%) cases of specific dermatoses of pregnancy. Raj et al.⁸³ reported specific dermatoses in 16.3% of cases. The specific dermatoses of pregnancy observed in our study was 14% (n = 70) and was seen exclusively in third trimester, which was statistically significant ($p < 0.0001$). Higher proportion of specific dermatoses was observed in primigravida (11.6%), which was statistically significant ($p < 0.0001$).

In literature, pruritus gravidarum is seen in the third trimester of pregnancy in 70% of cases⁵, with the incidence varying from 0.02% to 2.4% of pregnancies.²² Shivakumar and Madhavamurthy⁸¹ reported

3.52% and Raj et al.⁸³ reported 0.1% of cases of pruritus gravidarum. In our study, pruritus gravidarum was the most common specific dermatoses encountered (n =52, 10.4%). Of those affected, 44 (8.3%) were primigravidas, with the onset being in III trimester. The onset during third trimester is consistent with other studies.^{5, 81, 83}

The incidence of pruritic urticarial papules and plaques of pregnancy ranges between 1 in 130 pregnancies and 1 in 300 pregnancies.¹ Shivakumar and Madhavamurthy⁸¹ reported PUPPP in 2.35% of cases. Raj et al.⁸³ reported in 0.2% of cases. Kumari et al.² reported the occurrence of PUPPP in 14/22 cases. In our study, PUPPP was observed in 8 cases (8/70, 1.6%). Majority were primigravidas (n=7, 1.4%). All had single gestation pregnancy. No association with multiple gestation was observed in our study. The characteristic eruption occurred in all patients in III trimester. The parity (primigravida) and the onset in III trimester is consistent with other studies.^{2, 81, 85-87} Furthermore, the onset in III trimester and the primigravida being affected more was supported by the hypothesis that rapid abdominal wall distension in primigravidas may cause damage to connective tissue in the striae with conversion of nonantigenic molecules to antigenic ones, triggering an inflammatory response.^{21, 29-32}

Shivakumar and Madhavamurthy⁸¹, Raj et al.⁸³ and Kumari et al.² reported prurigo of pregnancy in 9.41%, 1.2% and 1/22 cases respectively. In this study, prurigo of pregnancy was seen in 1.8% (n=9) of cases.

Pruritic folliculitis of pregnancy was noticed in 0.2% (n=1), which is consistent with other studies.²

The incidence of pemphigoid gestationis is 1 in 50,000 pregnancies.³⁶ None of the patients in this study had pemphigoid gestationis.

The infections observed in our study was 30.8% (n=154). Statistically significant proportion of infections was observed in multigravidas (15.4%, p=0.0142).

The most common infection observed in our study was fungal infection, seen in 23.8% (n=119). Pityriasis versicolor was seen in 10.60% (n=53) of cases, the high prevalence of pityriasis versicolor may be attributed to warm humid environment in addition to the influence of pregnancy.⁵³

Shivakumar and Madhavamurthy⁸¹ reported candidiasis in 21.78% and Raj et al.⁸³ in 2.9%. In our study, Candidiasis was seen in 8% (n=40)

of cases. Candidal vulvovaginitis and intertrigo was observed in 34 cases (6.8%) and oral candidiasis in 6 patients (1.2%). It may be due to the higher glycogen content in the vaginal environment and estrogen mediated enhanced adherence of candida species to vaginal epithelial cells in pregnancy.⁵¹

In this study dermatophytosis was seen in 5.2% (n=26) of cases.

Bacterial infections observed in our study were 1.2%. Of the bacterial infections, furunculosis was seen in 0.8% of cases (n=4). One of them had gestational diabetes.

Hansen's disease was noticed in 0.4% (n=2) of cases. One was lepromatous leprosy and the other was borderline tuberculoid leprosy. Both the patients noticed the disease onset during pregnancy, which clearly shows the worsening of the disease during pregnancy, as described in literature.⁵⁵

Viral infections observed in our study were 4%. Shivakumar and Madhavamurthy⁸¹ reported condyloma acuminata in 4.70% of cases. In this study, warts were seen in 1.6% of cases (n=8), with 5 cases of condyloma acuminata (1%) and 3 cases (0.6%) of verruca vulgaris.

Condyloma acuminata in one of the patient had rapid growth during the course of pregnancy, which is in accordance with literature.⁵⁷

Herpes labialis and genital herpes were observed in 0.6% and 0.4% respectively in our study. This is consistent with other reports.⁸¹

Herpes zoster was observed in 1.2% (n=6), as pregnancy being an immunosuppressed state may act as a trigger factor for the development of herpes zoster.

The prevalence of HIV infection in pregnant women in India is about 0.3%.⁶¹ In our study, HIV was seen in 0.2% (n=1) of cases.

Trichomoniasis is seen in upto 60% of pregnant women.⁵ Shivakumar and Madhavamurthy⁸¹ reported 8.23% of cases of trichomoniasis. In our study, it was observed in 1.6% (n=8) of individuals. Scabies infestation was seen in 0.2% (n=1) in this study.

Acne vulgaris was observed in 10.4% (n=52) of individuals. 6.4% had onset of acne first during pregnancy and 4% had exacerbation of previous acne lesions, with most of the cases seen in III trimester (7.4%), as described in literature, this may be due to the increased sebum secretion rate due to a sebotrophic factor released from pituitary during the last trimester.¹⁵

Insect bite allergy was observed in 6% (n=30). This exaggerated response to insect bite allergy in pregnancy needs further study.

Acanthosis nigricans was seen in 0.4% (n=2) of cases. This may be a result of obesity.

Pityriasis rosea was seen in 0.4% (n=2).

Psoriasis was observed in 1 individual (0.2%), without any worsening during pregnancy, which is consistent with literature.⁷⁴

Impetigo herpetiformis was observed in 0.2% of cases (n=1) during III trimester. The onset of the disease is consistent with other studies.⁵ The patient was a known case of psoriasis vulgaris, who presented as generalized pustular psoriasis during pregnancy with typical features. As described in literature, this may be due to the hormonal influence of pregnancy over the disease, as an endocrine cause is being suspected for impetigo herpetiformis.³

Urticaria was seen in 0.2%, which needs further study.

Exacerbation of neurofibromatosis with increased number of cutaneous neurofibromas during pregnancy in III trimester was seen in 2 cases (0.4%). This is in accordance with literature, that neurofibroma may enlarge or arise denovo during pregnancy.⁵

Antiphospholipid antibody syndrome was noticed in 2 cases (0.4%). 3 cases (0.6%) of systemic lupus erythematosus was observed with cutaneous flares and arthritis, which is consistent with literature.⁶³

Comparison of this study report with other studies^{2,82,83}		
Conditions		Compared to other studies
Physiological changes	Pigmentary changes	Consistent
	Vascular changes	Consistent
	Striae gravidarum	Consistent
	Hair changes	Consistent
	Nail changes	Consistent
Pathological changes	Specific dermatoses	Consistent
	Pruritus gravidarum	Exceeds
	PUPPP	Consistent
	Prurigo of pregnancy	Exceeds
	Pruritic folliculitis	Consistent
	Candidiasis	Exceeds

In this study, the prevalence of physiological skin changes was much higher than specific dermatoses, stressing the fact that in most instances, the skin problems during pregnancy needs only reassurance. But meticulous observation and examination should be done, as pregnancy can influence many dermatological diseases and infections.

CONCLUSION

1. In this study, the prevalence of physiological skin changes in pregnant women was 94.8%, with pigmentary changes being the most common (90.8%), followed by striae gravidarum (79.6%), vascular changes (23.6%), miliaria (13.6%), hair changes (1.6%) and nail changes (0.8%).
2. The prevalence of specific dermatoses of pregnancy was 14%. Pruritus gravidarum was the most common disease encountered (10.4%), followed by prurigo of pregnancy (1.8%), pruritic urticarial papules and plaques of pregnancy (1.6%) and pruritic folliculitis of pregnancy (0.2%).
3. The prevalence of infections was 30.8%. The most common infection observed in our study was fungal infection (23.8%), followed by viral (4%) and bacterial (1.2%) infections. Pityriasis versicolor was the most common fungal infection observed (10.6%), followed by candidiasis (8%).
4. The miscellaneous dermatoses observed in this study in the order of prevalence were acne vulgaris (10.4%), insect bite allergy (6%), acanthosis nigricans (0.4%), pityriasis rosea (0.4%),

antiphospholipid antibody syndrome (0.4%), psoriasis (0.2%), impetigo herpetiformis (0.2%) and urticaria (0.2%). Exacerbation of systemic lupus erythematosus and neurofibromatosis during pregnancy was observed in 0.6% and 0.4% respectively.

5. Compared to multigravidas, primigravidas had a statistically significant proportion of pigmentary changes, striae gravidarum and specific dermatoses of pregnancy. Significantly higher proportion of vascular changes, striae gravidarum and specific dermatoses of pregnancy was observed in third trimester.

BIBLIOGRAPHY

1. Julie K Karen, Miriam Keltz Pomeranz. Skin changes and diseases in pregnancy. In : Klaus Wolff, Lowell A Goldsmith, Stephen I Katz et al. Fitzpatrick's Dermatology In General Medicine, 7th edn. Mc Graw Hill publications, 2008;955–62.
2. Kumari R, Jaisankar TJ, Thappa DM. A clinical study of skin changes in pregnancy. Indian J Dermatol Venereol Leprol 2007;73:141.
3. Millington GWM, Graham – Brown RAC. Skin and skin disease throughout life. In : Tony Burns, Stephen Breathnach, Neil Cox, Christopher Griffiths. Rook's Textbook of Dermatology, 8th edn. Wiley – Blackwell publications, 2010;8.9–13.
4. Cunningham FG, Kenneth J Leveno, Steven L Bloom et al. Implantation, embryogenesis and placental development. In: Williams Obstetrics, 23rd edn. Mc Graw Hill publications, 2009;36–77.
5. Kroumpouzos G, Cohen LM. Dermatoses of pregnancy. J Am Acad Dermatol 2001;45:1–19.
6. McKenzie AW. Skin disorders in pregnancy. Practitioner 1971;206:773–80.
7. Snell RS, Bischoff PG. The effects of large doses of estrogen and progesterone on melanin pigmentation. J Invest Dermatol 1960;35:73–82.
8. Winton GB, Lewis CW. Dermatoses of pregnancy. J Am Acad Dermatol 1982;6:977–98.
9. Sanchez NP et al. Melasma: a clinical, light microscopic, ultrastructural, and immunofluorescence study. J Am Acad Dermatol 1981;4:698–710.

10. McLeod SD, Ranson M, Mason RS. Effects of estrogens on human melanocytes in vitro. *J steroid Biochem Mol Biol* 1994;49:9–14.
11. Pathak MA, Riley FC, Fitzpatrick TB. Melanogenesis in human skin following exposure to long-wave ultraviolet and visible light. *J Invest Dermatol* 1962;39:435–43.
12. Lynfield YL. Effect of pregnancy on the human hair cycle. *J Invest Dermatol* 1960;35:323–27.
13. Trotter M. The activity of hair follicles with reference to pregnancy. *Surg Gynecol Obstet* 1935;60:1092–96.
14. Hellreich PD. The skin changes in pregnancy. *Cutis* 1974;13:82–6.
15. Burton JL, Shuster S, Cartlidge M. The sebotrophic effect of pregnancy. *Acta Derm Venereol* 1975;55:11–3.
16. Wong RC, Ellis CN. Physiologic changes in pregnancy. *J Am Acad Dermatol* 1984;10:929–40.
17. Demis DJ, Dobson RL, Mc Guire. *J Clinical Dermatology*. Vol 2. Unit 12 – 25. New York : Harper & Row : 1975;1–9.
18. Cummings K, Derbes VJ. Dermatoses associated with pregnancy. *Cutis* 1967;3:120–6.
19. Torgerson RR, Marnach ML, Bruce AJ, Rogers RS. Oral and vulvar changes in pregnancy. *Clin Dermatol* 2006;24:122–32.
20. Ambros – Rudolph et al. The specific dermatoses of pregnancy revisited and reclassified: Results of a retrospective two – center study on 505 pregnant patients. *J Am Acad Dermatol* 2006;54:395–404.

21. Kroumpouzou G, Cohen LM. Specific dermatoses of pregnancy: An evidence – based systematic review. *Am J Obstet Gynecol* 2003;188:1083–92.
22. Thappa DM, Srikanth Shanmugam. Pruritus gravidarum. *Ind J Dermatol* 1999;44(1).
23. Reyes H. Intrahepatic cholestasis : a puzzling disorder of pregnancy. *J Gastroenterol Hepatol* 1997;12:211–16.
24. Shornick JK. Dermatoses of pregnancy. *Semin Cutan Med Surg* 1998; 17:172–81.
25. Simon FR. Alterations of hepatic $\text{Na}^+ \text{K}^+$ ATPase and bile flow by estrogen effects on liver surface membrane lipid structure and function. *Proc Natl Acad Sci USA* 1978;75:4130–4.
26. Meng LJ, Reyes, H, Avelson M, Palma J et al. Progesterone metabolites and bile acids in the serum of patients with intrahepatic cholestasis of pregnancy: effect of ursodeoxycholic acid therapy. *Hepatology* 1997;26:1573–9.
27. Victoria Geenes, Catherine Williamson. Intrahepatic cholestasis of pregnancy. *World J Gastroenterol* 2009;15(17):2049–66.
28. Shaw D, Frohlich J, Wittmann BA, Willms M. A prospective study of 18 patients with cholestasis of pregnancy. *Am J Obstet Gynecol* 1982;142:621–5.
29. Cohen LM, Capeless EL, Krusinski PA, Maloney ME. Pruritic urticarial papules and plaques of pregnancy and its relationship to maternal – fetal weight gain and twin pregnancy. *Arch Dermatol* 1989;125:1534–6.

30. Bunker CB, Erskine K, Rustin MHA, Gilkes JJH. Severe polymorphic eruption of pregnancy occurring in twin pregnancies. *Clin Exp Dermatol* 1990;15:228–31.
31. Beckett MA, Goldberg NS. Pruritic urticarial papules and plaques of pregnancy and skin distention. *Arch Dermatol* 1991;127:125–6.
32. Powell FC. Parity, polypregnancy, paternity and PUPPP. *Arch Dermatol* 1992;128:1551.
33. Pawels C, Bucaille – Fleury L, Recanati G. Pruritic urticarial papules and plaques of pregnancy : relationship to maternal weight gain and twin or triplet pregnancies. *Arch Dermatol* 1994;130:80–2.
34. Campbell DM. Maternal adaptation in twin pregnancy. *Semin Perinatol* 1986;10:14–8.
35. Im S, Lee KS, Kim W, Song J, et al. Expression of progesterone receptor in human keratinocytes. *J Korean Med Sci* 2000;15:647–54.
36. Kristina semkova, Martin Black. Pemphigoid gestationis : Current insights into pathogenesis and treatment. *European Journal of Obstetrics & Gynecology and Reproductive Biology* 2009;145:138–44.
37. Morrison LH, Labob RS, Zone JJ, Diaz LA, Anhait GJ. Herpes gestationis autoantibodies recognize a 180 KDa human epidermal antigen. *J Clin Invest* 1988;81:2023–6.
38. Ortonne JP, Hsi BL, Verrando P, et al. Herpes gestationis factor reacts with the amniotic epithelial basement membrane. *Br J Dermatol* 1987;117:147–54.

39. Fairly JA, Heintz PW, Neuberger M, Diaz LA, Guidice GJ. Expression pattern of the bullous pemphigoid – 180 antigen in normal and neoplastic epithelia. *Br J Dermatol* 1995;133:385–91.
40. Lin MS, Gharia M, Fu CH, et al. Molecular mapping of the major epitopes of BP 180 recognized by herpes gestationis autoantibodies. *Clin Immunol* 1999;92:285–92.
41. Di Zenzo G, Calabresi V, Grosso F, et al. The intracellular and extracellular domains of BP 180 antigen comprise novel epitopes targeted by pemphigoid gestationis autoantibodies. *J Invest Dermatol* 2007;127:864–73.
42. Sitaru C, Mihai S, Zillikens D. The relevance of the IgG subclass of autoantibodies for blister induction in autoimmune bullous skin diseases. *Arch Dermatol Res* 2007;299:1–8.
43. Shornick JK, Jenkins RE, Briggs DC, et al. Anti HLA antibodies in pemphigoid gestationis. *Br J Dermatol* 1993;129:257–9.
44. Da Silva JA. Sex hormones and glucocorticoids : interactions with the immune system. *Ann N Y Acad Sci* 1999;876:102–18.
45. Hong Wu, Brian Schapiro, Terence J Harrist. Non infectious vesiculobullous and vesiculopustular diseases. In : David E Elder. *Lever's Histopathology of the skin*, 9th edn. Lippincott Williams & Wilkins publications, 2005;243–92.
46. Shornick JK, Black MM. Fetal risks in herpes gestationis. *J Am Acad Dermatol* 1992;26:63–8.
47. Chorzelski TP, Jablonska S, Beutner EH, Maciejowska E, Jarzabek – Chorzelska M. Herpes gestationis with identical lesions in the

newborn : passive transfer of the disease: Arch Dermatol 1976;112:1129–31.

48. Nurse DS. Prurigo of pregnancy. Australas J Dermatol 1968;9:258–67.
49. Roger D, Vaillant L, Fignon A, Pierre F, Bacq Y, Brechot JF, et al. Specific pruritic dermatoses of pregnancy : a prospective study of 3192 women. Arch Dermatol 1994;130:734–9.
50. Kroumpouzou G, Cohen LM. Pruritic folliculitis of pregnancy. J Am Acad Dermatol 2000;43:132–4.
51. Usha Gupta. Sexually transmitted diseases in pregnancy and neonate. In : Vinod K Sharma. A textbook of Indian association for the study of sexually transmitted diseases and AIDS, 2nd edn. Viva Publications, 2009;171–80.
52. Heymann WR, Wolf DJ. Malassezia (pityrosporum) folliculitis occurring during pregnancy. Int J Dermatol 1986;25:49–51.
53. Hay RJ , Ashbee HR. Mycology. In : Tony Burns, Stephen Breathnach, Neil Cox, Christopher Griffiths. Rook's Textbook of Dermatology, 8th edn. Wiley – Blackwell Publications, 2010;36.1–93.
54. Neena Khanna. Leprosy and pregnancy. In : Hemanta Kumar Kar, Bhushan Kumar. IAL Textbook of Leprosy, 1st edn Jaypee Brothers Medical Publications, 2010;313–24.
55. Jopling WH, McDougall AC. Pregnancy and Leprosy. In : Handbook of Leprosy. CBS Publishers and Distributors 1996;45–46.
56. Hastings RC, Oproimolk VA. Pregnancy and Leprosy. In : Leprosy. Longman Group UK limited 1994;284.

57. Sarla Malhotra. Sexually transmitted infections and pregnancy. In : Bhushan Kumar, Somesh Gupta. Sexually transmitted infections, 1st edn. Elsevier Publications, 2005; 885–96.
58. Singhal P, Naswa S, Marfatia YS. Pregnancy and sexually transmitted viral infections. Indian J Sex Transm Dis 2009; 30: 71–8.
59. Sterling JC. Virus infections. In : Tony Burns, Stephen Breathnach, Neil Cox, Christopher Griffiths. Rook's Textbook of Dermatology, 8th edn. Wiley Blackwell Publications, 2010; 33.1–81.
60. Enders G, Miller E, Cradock-Watson J et al. Consequences of varicella and herpes zoster in pregnancy. Prospective study of 1739 cases. Lancet 1994; 343: 1548–51.
61. Suneeta Mittal, Pakhee Aggarwal. HIV / AIDS in pregnancy. In : Vinod K Sharma. A textbook of Indian association for the study of sexually transmitted diseases and AIDS, 2nd edn. Viva Publications, 2009; 171–80.
62. Goodfield MJD, Jones SK, Veale DJ. The connective tissue diseases. In: Tony Burns, Stephen Breathnach, Neil Cox, Christopher Griffiths. Rook's Textbook of Dermatology, 8th edn. Wiley Blackwell Publications, 2010; 51.48.
63. Jill P Buyon, John Tamerius, Steve Ordorica, Bruce Young, Steven B Abramson. Activation of the alternative complement pathway accompanies disease flares in systemic lupus erythematosus during pregnancy. Arthritis and Rheumatism 1992; 35(1): 55–61.
64. Frances C, Piette JC. Cutaneous manifestations of Hughes syndrome occurring in the context of lupus erythematosus. Lupus 1997; 6: 139–44.

65. Miniati et al. Pregnancy in systemic sclerosis. *Rheumatology* 2008;47:16– 8.
66. Steen VD. Pregnancy in women with systemic sclerosis. *Obstet Gynecol* 1999;94:15–20.
67. Goodfield MJD, Jones SK, Veale DJ. The connective tissue diseases. In : Tony Burns, Stephen Breathnach, Neil Cox, Christopher Griffiths. *Rook's Textbook of Dermatology*, 8th edn. Wiley Blackwell Publications, 2010; 51.127.
68. Richard D Sontheimer, Melissa I Costner. Dermatomyositis. In : Klaus Wolff, Lowell A Goldsmith, Stephen I Katz et al. *Fitzpatrick's Dermatology in General Medicine*, 7th edn. MC Graw Hill Publications, 2008;1536–52.
69. Seema Chopra et al. Autoimmune inflammatory myopathy in pregnancy. *Medscape J Med* 2008;10(1):17.
70. Gutierrez G, Dagnino R, Mintz G. *Arthritis Rheum* 1984;27(3):291–4.
71. Adrian Perez Maldonado, Amal K Kurban. Metabolic diseases and pregnancy. *Clinics in Dermatology* 2006;24:88–90.
72. Marcia Ramos – e – Silva, Ana Libia Cardozo Pereira, Gustavo Bastos Oliveira, Sueli Coelho Da Silva Carneiro. Connective tissue diseases : PXE, anetoderma, and Ehler Danlos Syndrome in pregnancy. *Clinics in Dermatology* 2006;24:91–96.
73. Grin CM, Driscoll MS, Grantkels JM. The relationship of pregnancy, hormones and melanoma. *Semin Cutan Med Surg* 1998;17:167–71.

74. Oumeish Youssef, Abdul Wahab S Al Fouzan. Miscellaneous diseases affected by pregnancy. *Clinics in Dermatology* 2006;24:113–117.
75. Carlsten H, Nilsson N, Jonsson R, et al. Estrogen accelerates immune complex glomerulonephritis but ameliorates T cell – mediated vasculitis and sialadenitis in autoimmune MRL lpr / lpr mice. *Cell Immunol* 1992;144:190–202.
76. Clemens LE, Siiteri PK, Sites DP. Mechanism of immunosuppression of progesterone on maternal lymphocyte activation during pregnancy. *J Immunol* 1979;122:1978–85.
77. Mori T, Kobayashi H, Nishimoto H, et al. Inhibitory effect of progesterone and 20 alpha – hydroxypregn – 4 – en – 3one on the phytohemagglutinin - induced transformation of human lymphocytes. *Am J Obstet Gynecol* 1977;127:151–7.
78. Michelle Goldberg Green, Jennifer Bragg, Karla S Rosenman, Miriam Keltz Pomeranz. Pustular psoriasis of pregnancy in a patient whose dermatosis showed features of acute generalized exanthematous pustulosis. *Int J of Dermatology* 2009;48:299–303.
79. Beveridge GW, Harkness RA, Livingston JRB. Impetigo herpetiformis in two successive pregnancies. *Br J Dermatol* 1966;78:106–12.
80. Narciss Mobini, Sonia Toussaint, Hideko Kamino. Noninfectious erythematous, papular, and squamous diseases. In : David E Elder. *Lever's Histopathology of the skin*, 9th edn. Lippincott Williams & Wilkins Publications, 2005;179–214.
81. Shivakumar V, Madhavamurthy P. Skin in pregnancy. *Indian J Dermatol Venereol Leprol* 1999;65:23–5.

82. Muzaffar F, Hussain I, Haroon TS. Physiologic skin changes during pregnancy: A study of 140 cases *Int J Dermatol* 1998;37:429-31.
83. Raj S, Khopkar V, Kapasi A, et al. Skin in pregnancy. *Indian J Dermatol Venereol Leprol* 1992;58:84-88.
84. Martin AG, Leal Khouri S. Physiological skin changes associated with pregnancy. *Int J Dermatol* 1992;31:375-8.
85. Aronson IK, Halaska B. Dermatologic disease. In: Barron WD, Lindheimer MD, editors. *Medical Disorders During Pregnancy*, 2nd ed. St. Louis: Mosby, 1995:534-550.
86. Dotz W, Berman B. Dermatologic problems of pregnancy. In: Cherry SH, Merkatz IR, editors. *Complications of Pregnancy: Medical Surgical Gynaecologic Psychosocial and Perinatal*, 4th ed. Baltimore : Williams and Wilkins, 1991:562-587.
87. Black MM, Stephens CJM. The specific dermatoses of pregnancy: The British perspective. *Advances in Dermatology* 1991;7:105-126.

PROFORMA

Serial No

Name

Age

Address

Occupation

Income

Obstetric Status

LMP

EDD

Trimester

Antenatal H/O

Dermatological complaints

Onset of skin changes / lesions in relation to duration of pregnancy

Exacerbating / Relieving factors

H/O similar illness in previous pregnancies

Treatment H/O

Obstetric complaints

Any other skin / medical diseases

DERMATOLOGICAL EXAMINATION

A. PHYSIOLOGICAL

I. Hyperpigmentation

1. Linea nigra
2. Melasma
3. Diffuse pigmentation of areolae, nipples, abdomen and genitalia
4. Naevi

II. Vascular Changes

1. Gingival hyperplasia
2. Epulis of pregnancy
3. Varicose veins
4. Pedal edema
5. Abdominal wall edema
6. Capillary hemangioma
7. Spider angiomas
8. Palmar erythema

III. Connective tissue

1. Striae gravidarum
2. Acrochordons

IV. Changes in hair Growth

1. Increased hair growth

2. Hair loss

V. Eccrine Glands

1. Miliaria

VI. Nails

1. Brittleness of Nail Plate
2. Onycholysis
3. Others if any

B. DERMATOSES OF PREGNANCY

- I. Pruritus gravidarum
- II. Pruritic urticarial papules and plaques of pregnancy
- III. Prurigo of pregnancy
- IV. Pruritic folliculitis
- V. Pemphigoid gestationis

C. DERMATOSES AND TUMOURS MODIFIED BY PREGNANCY

I. Infections

1. Bacterial
2. Viral
3. Fungal
4. Protozoal
5. Parasitic infestations

II. Autoimmune disorder

1. Lupus Erythematosus
2. Systemic Sclerosis

3. Dermatomyositis / Polymyositis

III. Metabolic disorder

1. Porphyria cutanea tarda
2. Acrodermatitis enteropathica

IV. Disorders of Connective tissue

1. Ehlers – Danlos syndrome
2. Pseudoxanthoma elasticum

V. Tumors

VI. Miscellaneous

1. Acne vulgaris
2. Insect bite allergy
3. Psoriasis
4. Impetigo herpetiformis
5. Erythema multiforme
6. Others, if any.

INVESTIGATION

1. Scraping for fungus & KOH mount examination
2. Gram's stain
3. Tzanck smear
4. Slit skin smear for AFB
5. Blood sugar
6. Complete hemogram
7. Blood VDRL
8. ELISA for HIV
9. Liver function test for pruritus related diseases
10. Skin biopsy

11. Other relevant investigations

MASTER SHEET

[illegible]

KEY TO MASTER SHEET

OS	–	Obstetric status
TM	–	Trimester
LN	–	Linea nigra
ME	–	Melasma
PIG	–	Diffuse pigmentation
NV	–	Naevi
GH	–	Gingival hyperplasia
VA	–	Varicosity
PE	–	Pedal edema
AE	–	Abdominal wall edema
ST	–	Striae gravidarum
HA	–	Hair changes
NA	–	Nail changes
MR	–	Miliaria rubra
PrG	–	Pruritus gravidarum
PU	–	Pruritic urticarial papules and plaques of pregnancy
PP	–	Prurigo of pregnancy
PF	–	Pruritic folliculitis of pregnancy
PG	–	Pemphigoid gestationis
FU	–	Furunculosis
HD	–	Hansen disease
WA	–	Wart
HSV	–	Herpes simplex

HZ	–	Herpes zoster
HIV	–	Human immunodeficiency virus
CA	–	Candidiasis
TV	–	Tinea versicolor
DE	–	Dermatophytosis
TR	–	Trichomoniasis
SC	–	Scabies
AC	–	Acne vulgaris
IB	–	Insect bite allergy
AN	–	Acanthosis nigricans
PR	–	Pityriasis rosea
PS	–	Psoriasis
IH	–	Impetigo herpetiformis
SLE	–	Systemic lupus erythematosus
AP	–	Antiphospholipid antibody syndrome
NF	–	Neurofibromatosis
UR	–	Urticaria
+	–	Present
-	–	Absent

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு :

கருவுற்ற தாய்மார்களுக்கான சருமநோய்கள் பற்றிய ஆய்வு

ஆராய்ச்சி நிலையம் : சரும நலத்துறை மற்றும் மகப்பேறு மருத்துவமனை
அரசு பொது மருத்துவமனை,
சென்னை 600 003.

பங்கு பெறுபவரின் பெயர் :
பங்கு பெறுபவரின் எண். :
பங்கு பெறுபவர் இதனை (✓) குறிக்கவும்

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது.
என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை
பெறவும் வாய்ப்பளிக்கப்பட்டது.

☐

நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த
காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான்
இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.

☐

இந்த ஆய்வு சம்மந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும்
போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ
அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து
கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும்
என அறிகிறேன்.

☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும்
மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில்
பயன்படுத்திக் கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன்
சம்மதிக்கிறேன்.

☐

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட
அறிவுரைகளின்படி நடந்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும்
மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். என்
உடல்நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான
நோய்க்குறி தென்பட்டாலோ உடனே அதை மருத்துவ அணியிடம் தெரிவிப்பேன்
என உறுதி அளிக்கிறேன்.

☐

பங்கேற்பவரின் கையொப்பம் இடம் தேதி

கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம்

ஆய்வாளரின் கையொப்பம் இடம் தேதி

ஆய்வாளரின் பெயர்

INSTITUTIONAL ETHICAL COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-600 003.

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Dated .09.2009

L.Dis.No. 14597 / ME5/EthicsDean/MMC/2009

Title of the work
Principal Investigator

: A screening study on dermatoses in
pregnancy)

Dr. K. Kannambal . Ph - M.D (DVL)

Department

: Madras medical college - Ch-3.

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 23rd September 2009 at 2.00P.M. in Madras Medical College, Deans, Chamber, Chennai-3.

The members of the Committee, the Secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The principal investigator and their term are directed to adhere the guidelines given below:

1. You should get detailed informed consent from the patients/participants and maintain confidentiality.
2. You should carry out the work without detrimental to regular activities as well as without extra expenditure to the Institution or Government.
3. You should inform the IEC in case of any change of study procedure, site and investigation or guide.
4. You should not deviate from the area of the work for which I applied for ethical clearance.
5. You should inform the IEC immediately, in case of any adverse events or serious adverse reactions.
6. You should abide to the rules and regulations of the institution(s).
7. You should complete the work within the specific period and if any extension of time is required, you should apply for permission again and do the work.
8. You should submit the summary of the work to the ethical committee on completion of the work.
9. You should not claim funds from the Institution while doing the work or on completion.
10. You should understand that the members of IEC have the right to monitor the work with prior intimation.


SECRETARY
IEC, MMC, CHENNAI


CHAIRMAN
IEC MMC CHENNAI


DEAN
MADRAS MEDICAL COLLEGE
CHENNAI